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THIRD SERIES FOUNDED BY EDWARD JACKSON

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DEPARTMENTS

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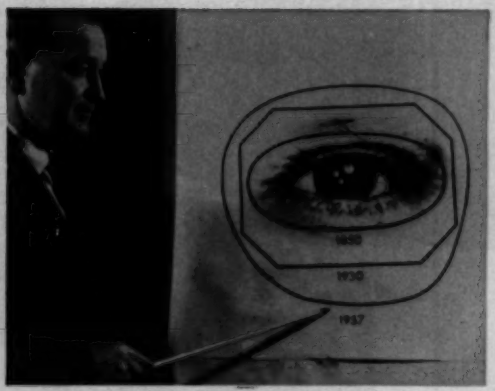

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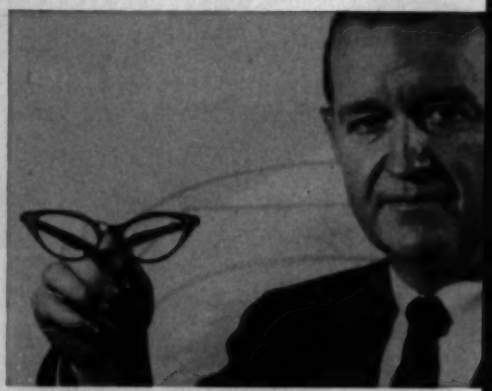

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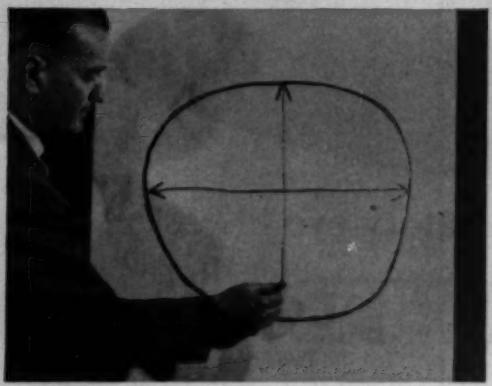

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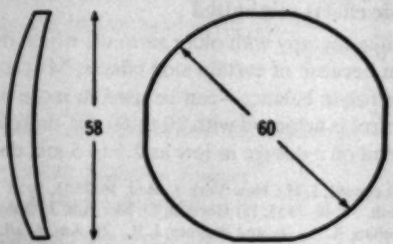

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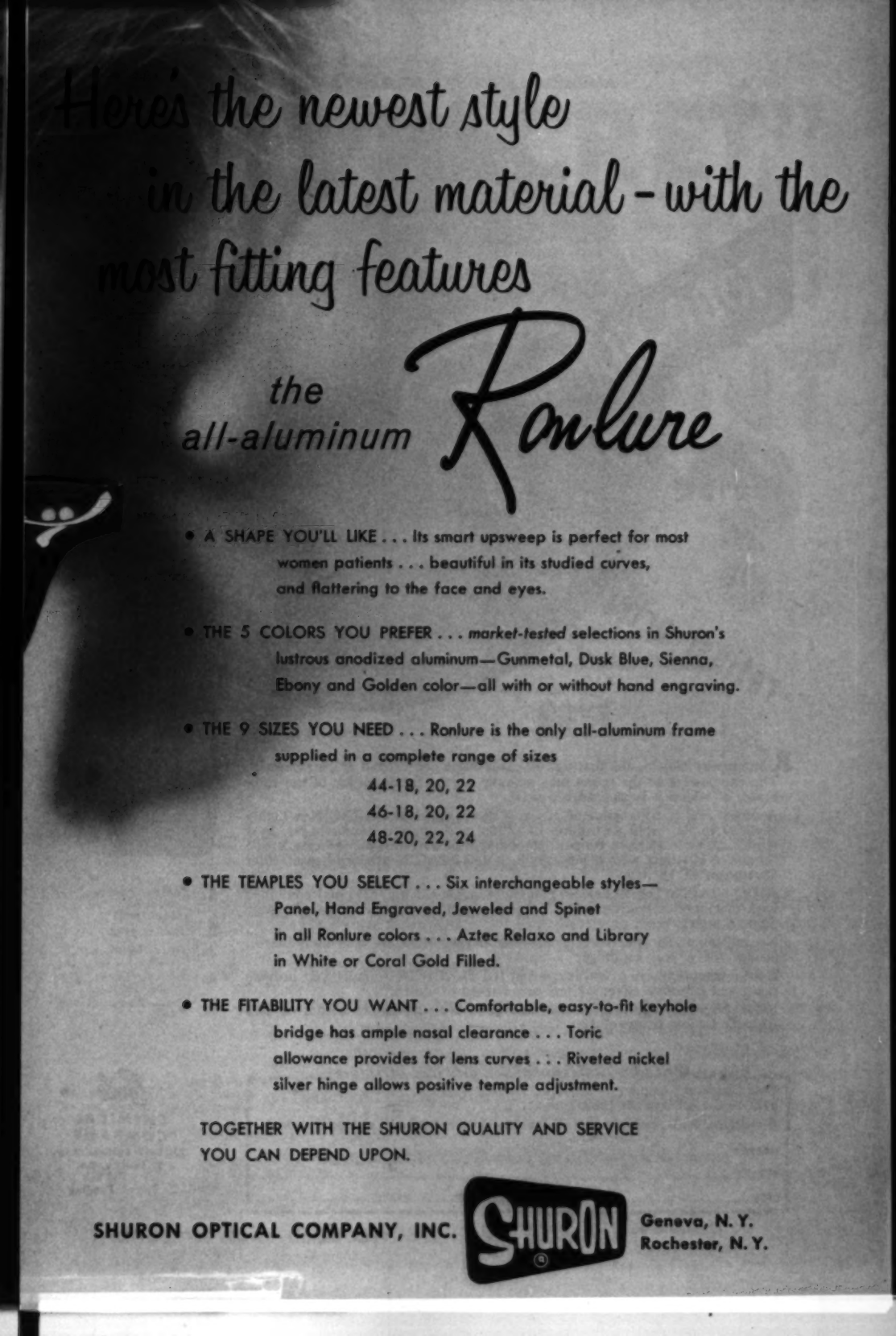
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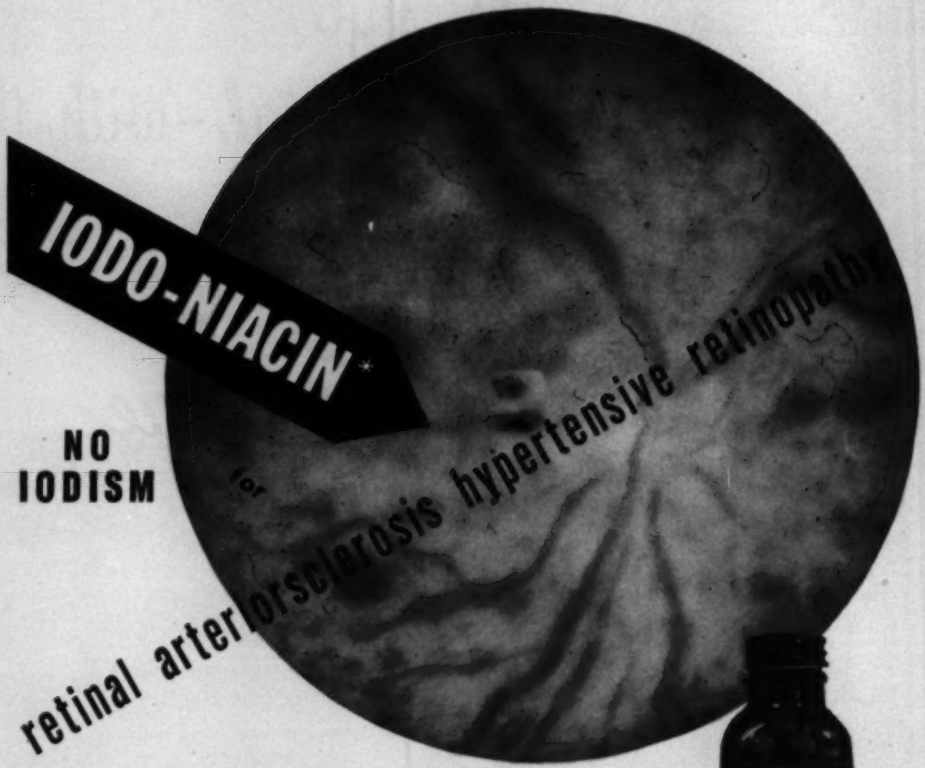

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1. *Cecil's Textbook of Medicine*, 7th ed., 1947, p. 1287.
2. *Ibid.*, p. 1598.
3. *Am. J. Ophth.* 42:771, 1956.
4. *Am. J. Digest. Dis.* 22:5, 1955.
5. *Med. Times* 84:741, 1956.

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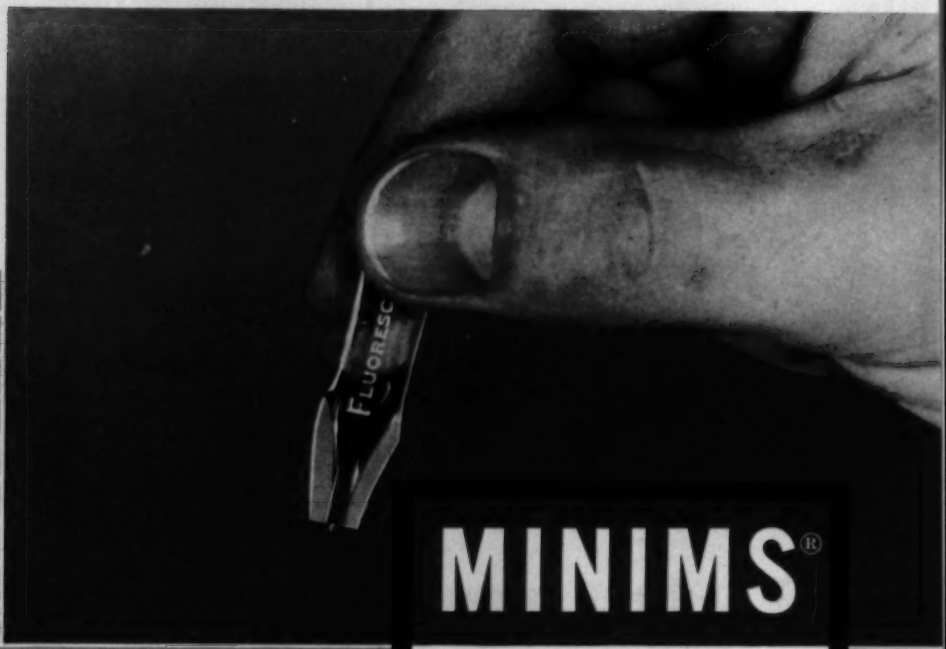

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AMERICAN JOURNAL OF OPHTHALMOLOGY

VOLUME 45 APRIL, 1958 NUMBER 4, PART I

TUMORS OF THE RETINAL PIGMENT EPITHELIUM*

JOHN R. FAIR, M.D.
Augusta, Georgia

In the study of intraocular neoplasms, a particularly profitable exercise is an excursion into the field of those uncommon new growths, the epithelial tumors of the inner eye. The rarity of these tumors makes them of little clinical significance but some insight into their origin and behavior adds immensely to our understanding of the more frequently encountered retinoblastoma and malignant melanoma.

In embryonic life, the outer and inner layers of the secondary optic vesicle are the forerunners of the intraocular epithelial structures—the anterior and posterior epithelial layers of the iris, the pigmented and unpigmented layers of the ciliary epithelium, and the pigment epithelium of the retina (fig. 1). These epithelial elements maintain their original simple state while the optical portion of the retina is developing its complex form. The fact that the sensory retina gives rise to tumors much more frequently than either the primitive iris or ciliary epithelium may be accounted for by the power of differentiation inherent in retinal cells. Whether or not this is true, primary intraocular epithelial tumors are extremely rare. The ciliary epithelium has produced the majority of the reported cases, the iris being responsible for the remainder. True tumors of the retinal pigment epithelium are almost unknown.

Despite the small number of cases involved, epithelial tumors have attracted considerable attention in the past. Some are found incidentally in eyes removed for unrelated causes. Others arise in response to severe and prolonged inflammation of the inner eye. One special type originating in the ciliary epithelium attempts to form embryonic retina. This paper is concerned with tumors of the retinal pigment epithelium. The author of a recent report on the same subject would limit the number of authentic cases of this kind to three, all of which arose incidentally in eyes which were the site of a long standing and severe inflammation. A careful perusal of the literature, however, reveals at least four additional examples, one of which occurred, apparently, in an otherwise normal eye. It is the purpose of this study to present a new case of primary epithelioma of the retinal pigment epithelium and to point out the close resemblance of these tumors to those of the iris and ciliary epithelium.

CASE REPORT

A white woman, aged 26 years, was first seen October 11, 1951, complaining of progressive loss of vision in the left eye which had begun three years before. Prior to that time there had been no ocular complaints and the patient had been well generally. There was no history of ocular injury.

In February, 1948, during a routine examination, there was found a diminution of vision in the left eye. This examination was done elsewhere and, according to the ophthalmologist who saw the patient at that time, "revealed a large lesion between the macula and the nerve head of the left eye. The retina was thickened and edematous in this region with the beginning of a star figure at the macula. The underlying pigment epithelium was heavier than in the surrounding area." The examiner's impression was "that the best possibility was an inflammatory lesion in the choroid adjacent to the nerve head with edema of the overlying retina."

Progress studies were advised because the examination "did not rule out the possibility of a neoplasm." On May 17, 1948, the lesion was de-

* From the Division of Ophthalmology, Department of Surgery, Medical College of Georgia.

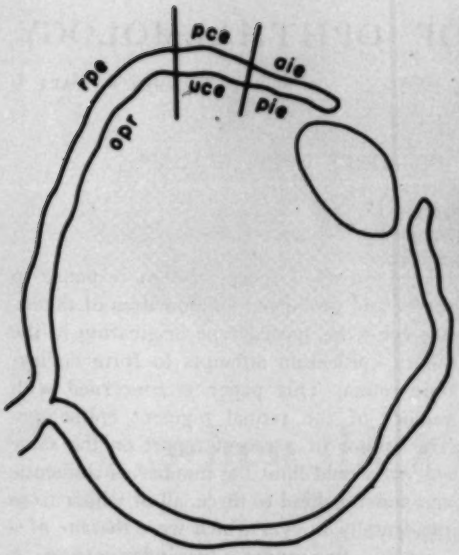

Fig. 1 (Fair). Diagram of eye during its early development to show origin of epithelial layers of iris, ciliary body, and retina. (aie) Anterior layer of iris epithelium. (pie) Posterior layer of iris epithelium. (pce) Pigmented ciliary epithelium. (uce) Unpigmented ciliary epithelium. (rpe) Retinal pigment epithelium. (opr) Optical portion of retina.

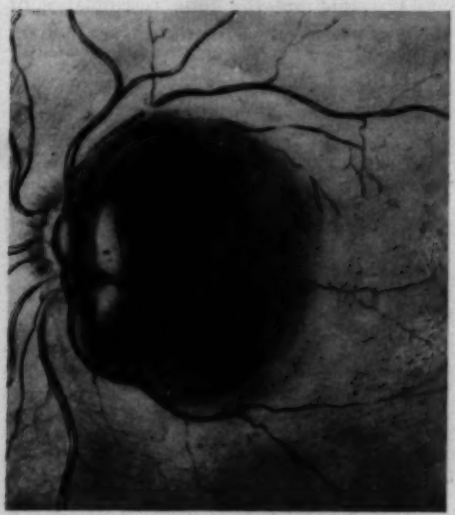

Fig. 2 (Fair). Drawing of left fundus made in 1949 to show pigmented swelling between nerve head and macula.

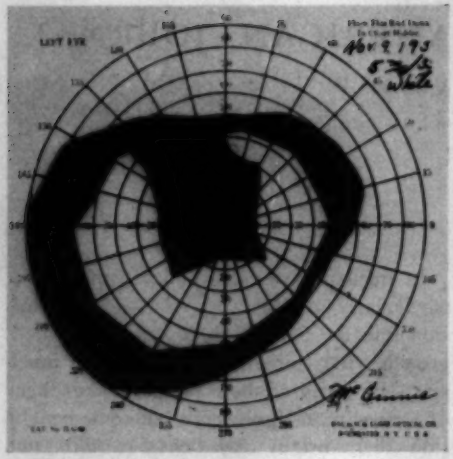

Fig. 3 (Fair). Extent of visual field which remained at time of enucleation.

scribed as being a little larger and showed definite exudative reaction in the overlying vitreous. The possibility of tumor was still considered. Figure 2 is a drawing of the fundus made in 1949.

In the following two years the patient did not consult an ophthalmologist, although there was a steady decrease of vision in the left eye. In October, 1951, only hand movements remained. The vision in the right eye was recorded as 20/30 correctible to 20/15—1. External examination revealed no abnormality of either eye. Ocular tension was 20 mm. Hg (Schiøtz), bilaterally. Ocular rotations were normal and the eyes were straight for both distance and near. In the right eye the media were clear and the fundus normal as seen through the widely dilated pupil. In the left eye the media were clear. The left fundus contained a massive, smoothly rounded, dark gray swelling which arose at the posterior pole and obscured the nerve head and the central area of the retina. The tumor had a solid appearance. The overlying retina was intact. The summit of the mass was elevated six diopters. Its sides met the surrounding retina at a sharp angle. Along the periphery of the base were small collections of white material. No hemorrhages were seen. The unaffected retina between the tumor and the equator was normal in appearance. It was impossible to photograph the lesion because of its size. Figure 3 indicates the field of vision which remained.

General physical examination revealed no abnormalities and routine laboratory studies were within normal limits, as were X-ray films of the chest and skull.

It was the opinion of numerous ophthalmologists that the tumor was a malignant melanoma and enucleation was recommended. This procedure was carried out November 20, 1951. Subretinal fluid

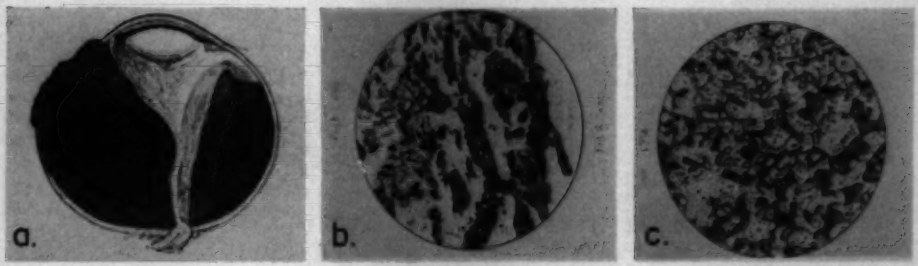

Fig. 4 (Fair). Copy of drawings from report of melanotic malignant tumor of inner eye by Griffiths, in 1894. (a) Gross specimen. (b) High-power microscopic view. (c) Low-power view.

aspirated at the time of enucleation showed "cells consistant with malignancy." The patient's recovery was uninterrupted and in the ensuing five years there has been no recurrence of the growth. Pathologic examination* of the enucleated globe was reported as follows:

Gross. The specimen consists of a partially collapsed eye measuring 24 by 23 by 21.5 mm. There is a small hole in the sclera near the equator at the site of puncture. The eye is opened in the horizontal plane. A pigmented mass measuring 4.5 by 2.5 by 7.0 mm., and gelatinous material containing small opacities are present beneath the retina in the macular region. The mass obscures the nerve head.

Microscopic. The anterior segment is not remarkable. Adjacent to the nerve head is a mass composed of cells derived from retinal pigment epithelium. The cells are moderately but not uniformly pigmented and, for the most part, have proliferated in the characteristic tubular manner, although in some areas the arrangement is diffuse. They have invaded the choroid, the overlying retina, and the nerve head. The nerve head has been displaced laterally and that half of it which is not invaded is edematous. There is columnar gliosis of the optic nerve on the side of the mass. The adjacent retina is detached with serous exudate containing clumps of pigment-laden cells beneath it. Mild chronic inflammatory cell infiltration is present in the choroid around the tumor and there is lymphocytic periphlebitis in the nerve head. This suggests that, as sometime occurs in massive proliferations of retinal pigment epithelium, the tumor may have arisen in an area of long standing chronic inflammation. However, because of local invasion, it must be regarded as a true neoplasm rather than as a pseudoepitheliomatous hyperplasia. The possibility that the inflammatory reaction is secondary to the neoplasm cannot be ruled out. No proven case of metastasis from any type of growth having its origin in the pigment epithelium of the retina has been reported.

* Histopathologic examination performed at the Armed Forces Institute of Pathology, Washington, D.C.

Background

The first mention of the possibility of tumors of the retinal pigment epithelium was made by Griffith[1] in 1894 when he reported a melanotic tumor of the inner eye (fig. 4) the cells of which were epithelial in appearance and grew in intercommunicating tubes and columns. Griffith felt certain that the growth arose from the pigment epithelium of the retina but because the tumor recurred quickly in the orbit and brought about the death of the patient, subsequent observers[2,3] are inclined to believe that the case in reality was that of a malignant melanoma. If this interpretation is correct, it is not because Griffith's powers of observation and description were lacking but only because the intervening years have taught us that epithelial tumors of the inner eye do not tend to recur locally or to metastasize.

In 1902, Alt[4] called attention to his past writings on the subject of intraocular epithelial new formations in which he described the proliferation of cells of the "uveal layer" in the shape of cylindric tubes which grew into cyclitic membranes and gave off branches. Alt believed that each of the two layers of the ciliary epithelium might form tumors characterized by tubes of cells and that very similar changes took place in the "pigment epithelium of the choroid" (retinal pigment epithelium). In this work, Alt described two instances of proliferation of the retinal pigment epithelium into tumorlike masses, one in the case of an eye injury (fig.

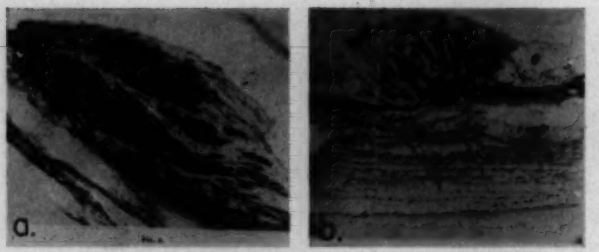

Fig. 5 (Fair). Copy of photographs from Alt's report on proliferation of retinal pigment epithelium in 1902. (a) Case of eye injury. (b) Case of retinal detachment.

5-a) and the other in connection with a retinal detachment (fig. 5-b).

In a lengthy discussion of proliferations and tumors of the ciliary epithelium, E. Fuchs[5] in 1908 mentioned a case of long standing intraocular inflammation in which there arose multiple tumors consisting of cell tubes from both layers of the ciliary epithelium and from the pigment epithelium of the retina (fig. 6). Fuchs classified these growths as malignant because of their local invasiveness.

Deutschmann[6] in 1912 described a glioma of the retina (retinoblastoma) which he felt arose from the pigment epithelium. In 1915, Reis[7] disagreed with Deutschmann and presented a similiar case to show that gliomas growing between choroid and retina might be mistaken as to their site of origin.

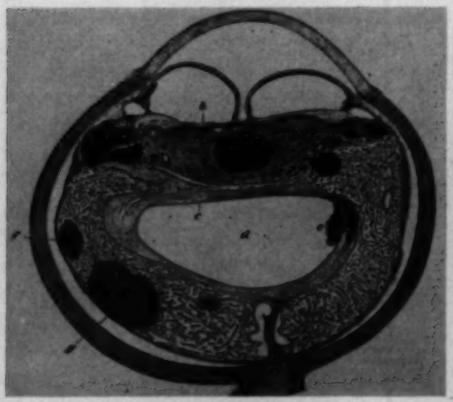

Fig. 6 (Fair). Copy of drawing of case reported by Fuchs in 1908. A central cavity is surrounded by a thick membrane containing bone. Numerous pigmented epithelial tumors are seen.

Proliferation of the retinal pigment epithelium following extirpation of a tumor of the optic nerve was described by Koyanagi[8] in 1913. The eye, which was removed sometime later, showed a remarkable overgrowth of the pigment epithelium. In the region of the disc, tubular columns that resembled glandular tissue extended deep into the nerve (fig. 7). Koyanagi did not use the term "tumor" in connection with this case.

In 1921, v. Maertens[9] reported a malignant epithelial tumor of the inner eye which he felt was similar to that described by Fuchs in 1908. This was the case of a 46-year-old woman in whom one eye had been blind since childhood. A tumor which arose in the

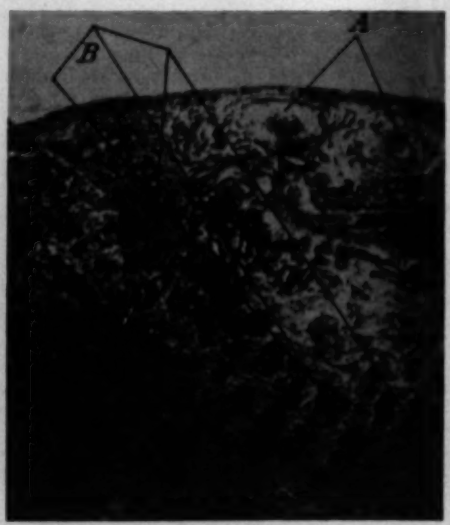

Fig. 7 (Fair). Copy of illustration of Koyanagi's case reported in 1913.

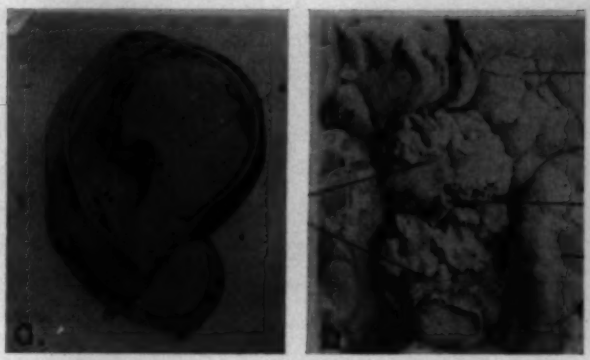

Fig. 8 (Fair). Copy of illustrations, showing gross specimens, in case reported by v. Maertens in 1921. (a) Eye filled with tumor. (b) Base of brain to show extension of tumor.

eye (fig. 8-a) spread by way of the optic nerve to the base of the brain (fig. 8-b) and brought about the patient's death. Autopsy showed no tumor elsewhere in the body from which the ocular neoplasm might have originated. Figure 9 shows other illustrations from this case. The passage of the years makes this tumor difficult to evaluate. v. Maertens was convinced that the new growth arose from ciliary or pigment epithelium or both.

Koyanagi[10] in 1938 reported a pigment-forming membranous tumor arising from the pigment epithelium of the retina in a case of bronchogenic carcinoma metastatic to the choroid. According to the author, this tumor did not show tubule formation but instead grew in a membrane over the outer surface of the detached retina (fig. 10).

Several tumors of the retinal pigment epithelium have been reported in recent years. Rønne[11] has described briefly a locally invasive growth which arose in an otherwise normal eye. This was a heavily pigmented tumor which was thought clinically to be a malignant melanoma. On removal of the eye, there was found a tumor arising from the pigment epithelium of the retina which had broken through the lamina vitrea into the choroid. There was no evidence of injury or inflammation. Rønne's description leaves little doubt as to the authenticity of this case.

Hyperplasia of the pigment epithelium of the retina simulating a neoplasm in an eye which was the site of a long standing inflammation was reported by Stow.[12] Stow did not use the term "tumor" in describing this case. Although the growth reached neoplastic proportions, local invasion was not seen. However, the cellular details of the mass (fig. 11)—large polygonal cells with pale staining oval nuclei arranged in cords, tubes, and sheets—are exactly the same as those in the cases of Fuchs, v. Maertens, Alt, and the case presently being described.

In his textbook,[2] *Tumors of the Eye*, Reese describes a tumorlike mass arising from the pigment epithelium of the retina in a case of long drawn out inflammation of the inner eye (fig. 12). Cellular details are not mentioned but it was felt by all who studied the sections that this was an example of pure hyperplasia.

In his discussion of tumors arising in the pigment epithelium of the iris, Laval[13] mentions a case seen by Dr. M. Matusow in which a pigmented mass in the fundus of an only eye was suspected of being a proliferation of retinal pigment epithelium because of its proximity to an old healed chorioretinitis.

The latest report of a tumor of the retinal pigment epithelium is that of Greer.[14] His case again is that of a chronically inflamed eye in which there was found a small tumor made up of large unpigmented polygonal

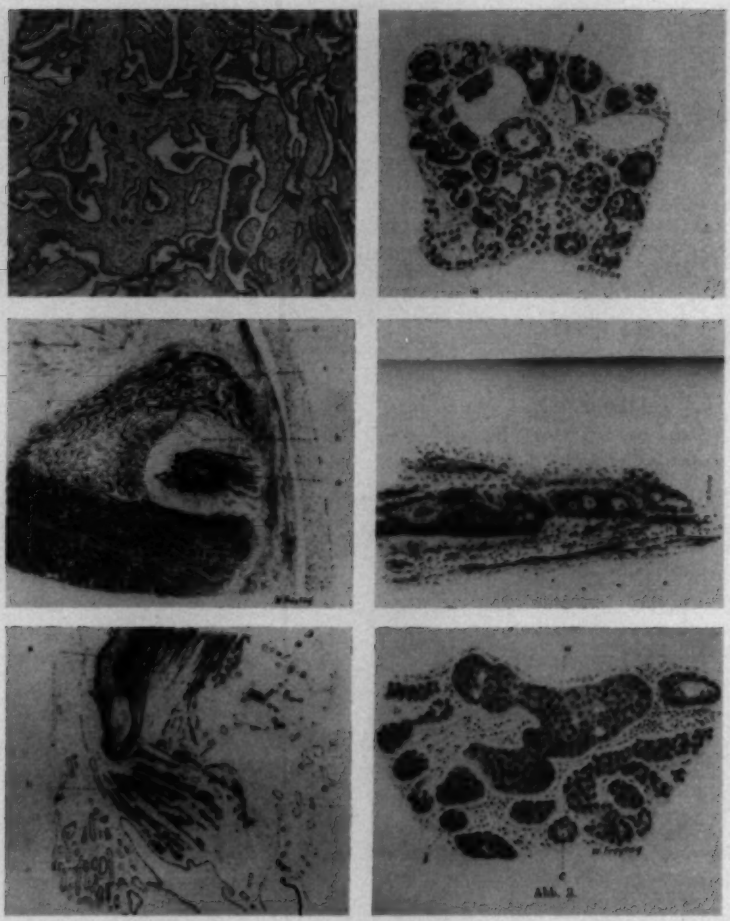

Fig. 9 (Fair). Copy of illustrations from v. Maertens' case, showing microscopic appearance of tumor.

cells based upon and arising from the pigment epithelium of the retina (fig. 13). The tumor had invaded the overlying retina but was not considered malignant because mitoses were very scanty, the cells appeared quiescent, and the mass was reasonably circumscribed. Greer felt that growths of this kind should be arranged upon a graduated scale at one extreme of which lie the obvious hyperplasias of inflammatory origin in which active inflammatory processes are dominant and, at the other extreme, the obvious epithelial neoplasms. One reason for the present study was to search out additional cases for comparison including tumors arising in otherwise normal eyes and another to show that "primary" and "secondary" growths of this kind are alike microscopically not only to themselves but also to similar neoplasms of both iris and ciliary epithelium.

Discussion

In considering the case which prompted this report, three questions immediately arise. First of all, one must decide whether or not the growth is a true tumor. The

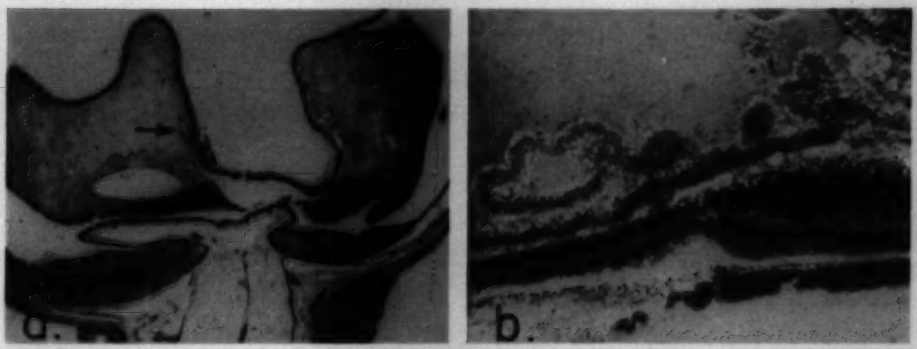

Fig. 10 (Fair). Copy of illustrations from case reported by Koyanagi in 1938, showing membranous tumors growing along outer surface of detached retina. (a) The metastatic tumor growing in the choroid. The small arrow indicates the second tumor. (b) Microscopic appearance of second tumor arising from retinal pigment epithelium.

retinal pigment epithelium is well known for its power of proliferation. It may be observed to fill in large defects in the retina caused by localized inflammation or injury. The distinction between hyperplasia and neoplasia is sometimes difficult to draw. Here, however, local invasiveness (fig. 14-b) and the presence of an occasional abnormal mitotic figure (fig. 14-f) tend to support the feeling of all who have studied sections of the growth that it represents a true tumor.

There is no apparent inflammatory change which might have stimulated proliferation of the pigment epithelium. Although a "mild chronic inflammatory cell infiltration" was described by the original examiner of the histopathologic section, the inflammatory changes are so slight that they might easily have been stimulated by the tumor itself. Figure 14-e shows the absence of inflammatory cells in the choroid adjacent to the growth. I believe that this answers satisfactorily the second problem—whether the tumor arose spontaneously or was secondary to chronic inflammatory disease. Other examiners interested especially in ocular pathology and ocular tumors who have studied this case are of the same opinion. The ophthalmologist who first described the le-

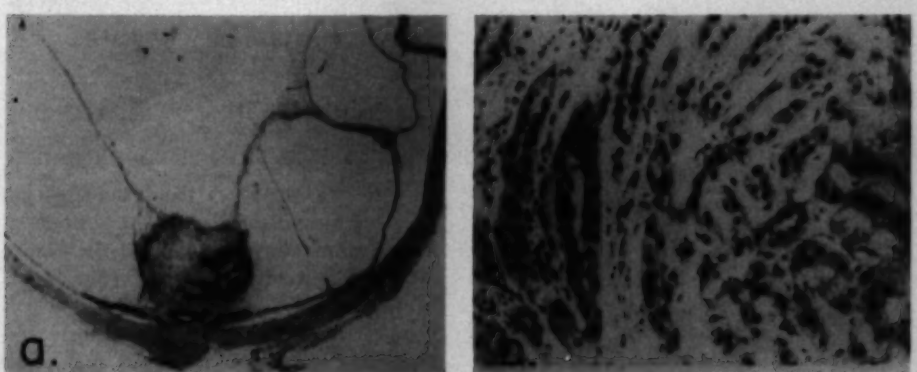

Fig. 11 (Fair). Copy of illustrations from Stow's report on hyperplasia of the pigment epithelium in 1949. (a) Position of growth at posterior pole. (b) Microscopic appearance.

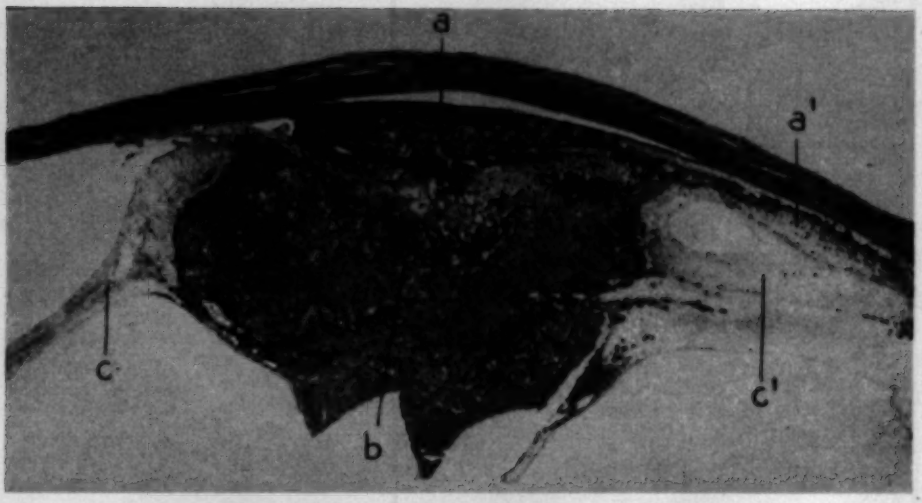

Fig. 12 (Fair). Reproduction of illustration in Reese's *Tumors of the Eye* (New York, Hoeber, 1951), showing tumorlike mass of proliferated retinal pigment epithelium.

sion clinically felt that an inflammatory process was the best possibility because of edema of the overlying retina and "exudative reaction" in the vitreous. His impression cannot be ignored but the histopathologic picture certainly suggests that inflammation was not an important etiologic factor.

The third question involves the malignancy of the tumor—or its lack of malignancy. While a number of the cases reported in the past have shown local invasiveness, none are known to have metastasized unless v. Maertens' case is seriously considered. None are known even to have breeched the scleral coat of the eye. Generalizations cannot be made use of in determining the malignancy of a new growth. While local invasiveness is one of the criteria of malignancy, it is not enough alone to support a diagnosis of malignancy in this case. Its benign course, the regular arrangement of its cells, the uniformity of the nuclei, and the infrequency of mitotic figures, all speak against a malignant nature. It is best, perhaps, to leave the question of malignancy unsettled until more cases present themselves for study.

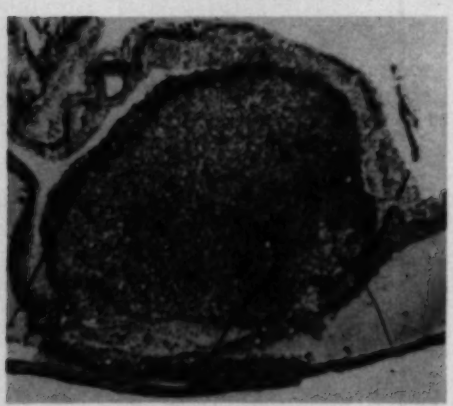

Fig. 13 (Fair). Copy of illustration in Greer's report of 1952, showing small tumor of retinal pigment epithelium.

Figure 14 illustrates the histopathologic characteristics of the lesion. The position of the growth in the eye is shown in Figure 14-a. Invasion of the choroid (fig. 14-b), the familiar cords and tubes of cells which characterize these tumors (fig. 14-e), and the sheets of cells seen in this and other examples (fig. 14-d) are depicted.

Of all those described in the past, the

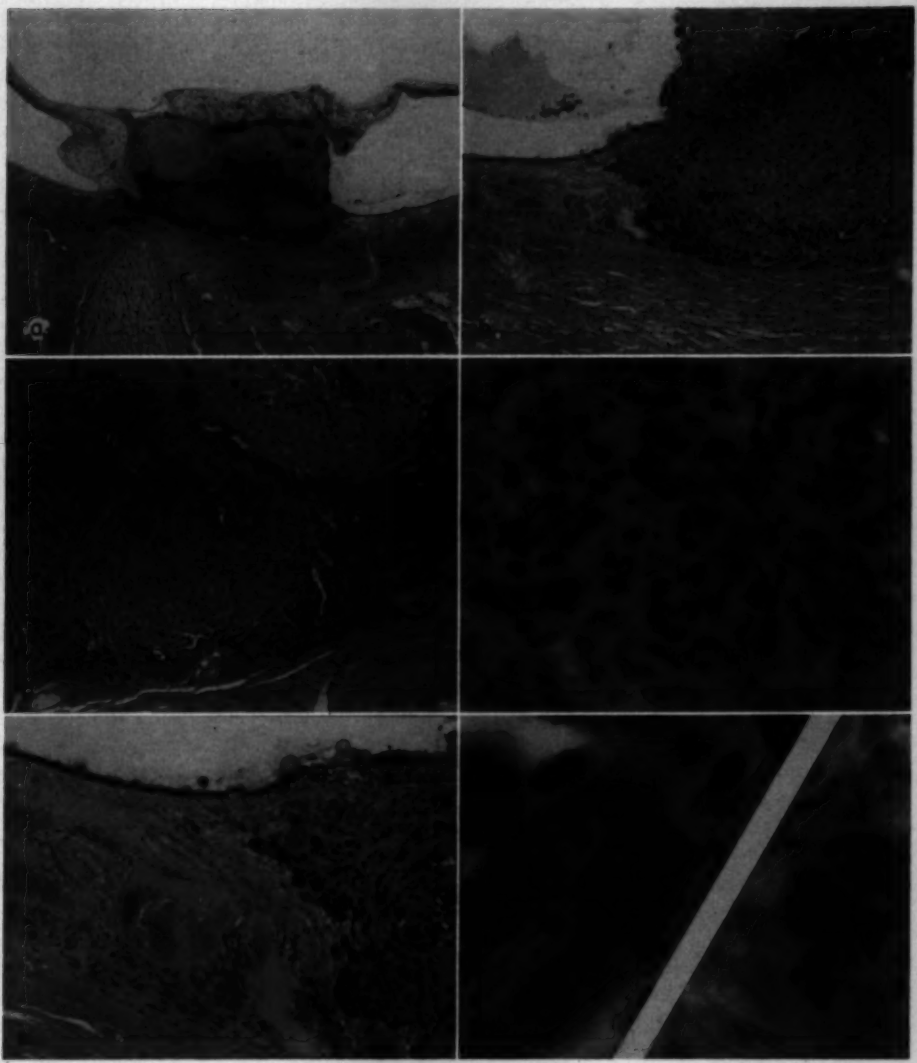

Fig. 14 (Fair). Microscopic appearance of the tumor which is the subject of this report. (a) Low-power view to show position of tumor at posterior pole of the eye. Invasion of the nerve head, choroid, and overlying retina is obvious. (b) Edge of tumor invading choroid. (c) Characteristic tubes and cords of cells which make up the tumor. (d) Portion of tumor in which the arrangement of the cells is more diffuse. (e) Few or no inflammatory cells are seen in the choroid adjacent to the tumor. (f) Rare normal and abnormal mitotic figures.

present case most closely resembles that of Rønne. Each arose in an otherwise normal eye in contrast to those tumors originating in chronically inflamed eyes or eyes that were the site of other abnormal changes such as a metastatic tumor (Koyanagi, 1938) or degenerative process (Koyanagi, 1913).

The fact that chronic inflammation may

stimulate the epithelial layers to proliferation and even tumor formation is well known but unexplained. Whatever the reason for this phenomenon, it is quite obvious that there is no difference between the tumors that arise in chronically inflamed eyes and those that occur spontaneously as did the present case and that of Rønne. In each situation, there are the same large epithelial cells with oval nuclei arranged in the characteristic pattern of tubes and cords with pink-staining homogenous material between. Pigmentation differs from case to case and between different areas of the same tumor. The only variation is in the presence or absence of local invasion of adjacent structures, an important distinction, perhaps, in determining the activity of the individual growth.

An even more important consideration is the similarity between tumors and proliferations of the retinal pigment epithelium and those of the epithelial layers of the iris and ciliary body. The cells of these tissues have a common origin and are much alike in their final state so, as might be expected, they react alike to injury and inflammation and produce neoplasms of the same type. It is only in recent years that a sufficient number of tumors of the retinal pigment epithelium has appeared to make this similarity apparent. Comparison will show the same histopathologic characteristics in all.

Each example of these rare tumors recorded increases the importance of their nomenclature and classification. The original scheme for classifying tumors of the ciliary epithelium devised by Fuchs at the turn of the century was based upon a very small number of cases. Since then there have been described tumors which combine the features of his "diktyoma" and "malignant epithelioma" and, in addition, tumors of the iris epithelium have been shown to simulate closely those of the ciliary body. Indeed, a "diktyoma" of the optic nerve has recently been reported by Reese.[15]

It has been suggested by others[16, 17] that the term "medullo-epithelioma" be used in describing tumors of the epithelium of the ciliary body and iris and that these growths be further divided into those of "embryonic type" (diktyoma of Fuchs) and "adult type" (malignant epithelioma). The same reasoning should be and is applied to the retinal pigment epithelium since it too originates as primitive medullary epithelium of the neural tube. All tumors of the retinal pigment epithelium reported to date have been of the adult type as is the case being presented, its only unusual feature being the fact that it apparently arose spontaneously in an otherwise normal eye.

Summary

1. A new case of medullo-epithelioma arising in the pigment epithelium of the retina is presented. The tumor occurred spontaneously as nearly as can be told.

2. The literature on the subject is reviewed and it is pointed out that tumors of the retinal pigment epithelium are similar or identical to those produced by the epithelium of the iris and ciliary body.

Ophthalmology Division.

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ARCUATE DEFECTS IN THE VISUAL FIELDS DUE TO CHROMOPHOBE ADENOMA OF THE PITUITARY GLAND*

CLINICS IN PERIMETRY, No. 1

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Bitemporal hemianopsia, the classic perimetric feature of chromophobe adenoma of the pituitary gland, usually does not present a great diagnostic problem. If the patient is reasonably co-operative such defects may be plotted accurately and the examiner is able to interpret them as being diagnostic of a chiasmal lesion of some type.

Occasionally, chromophobe adenomas produce arcuate defects in the visual fields and even the most experienced perimetrist will be led astray in the diagnosis unless he is aware of this possibility. Arcuate defects generally are characteristic of lesions of the retina or anterior portion of the optic nerve and suggest glaucoma, closure of an arteriole in the retina or optic nerve, hyaline bodies in the nervehead, choroiditis, or other retinal or optic nerve lesions. Ophthalmoscopic examination usually provides an explanation for the defects. If the cause for the arcuate defect is not obvious with the ophthalmoscope, the examiner is likely to assume that there has been closure of a small arteriole in the anterior portion of the optic nerve which is not visible. Such reasoning, although sometimes correct, may be a diagnostic pitfall as the following cases demonstrate.

REPORT OF CASES

CASE 1

A 61-year-old man, first seen at the Mayo Clinic on March 27, 1957, complained of gradually failing vision in the left eye of one and one-half years' duration. He had also had some ache on the left side of his head for about the same length of time which had become worse over the last six months.

Examination of the eyes showed opacities in the lower nasal sectors of the lenses of both eyes, the greater on the right, which seemed to explain the poorer central acuity of this eye. The right optic disc had a good pink color while the left disc had pallor, grade 2. There was no cupping of either disc and the intraocular pressure measured 14 mm. Hg (Schiøtz) in each eye.

The visual fields (fig. 1) showed a dense inferior arcuate defect in the left eye while the field of the right eye contained a minimal superior temporal depression. A roentgenogram of the head disclosed an enlarged sella turcica with erosion of the dorsum, deepening of the floor, and erosion of the left anterior clinoid due to either an intrasellar or a parasellar tumor. Transfrontal craniotomy revealed a large cystic tumor which the sur-

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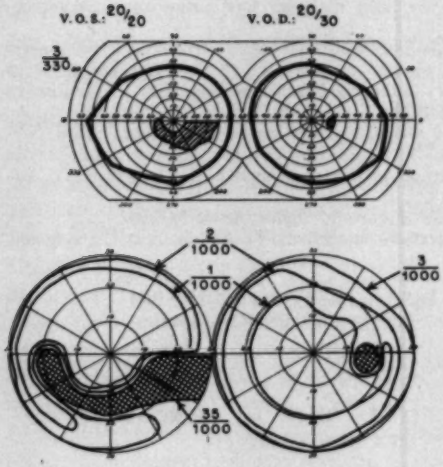

Fig. 1 (Kearns and Rucker). *Case 1.* Dense inferior arcuate defect in the field of the left eye due to a chromophobe adenoma of the pituitary gland.

gical pathologist identified as a chromophobe adenoma of the pituitary.

CASE 2

A 61-year-old man was seen in the Section of Ophthalmology on June 28, 1956, because of blurred vision of the left eye over the previous two to three months. The right eye had good central acuity but the left eye could not be improved by refraction beyond 20/30. The right disc had a minimal pallor; the left disc had a mild but definite pallor. The intraocular pressure was 20 mm. Hg (Schiøtz) in each eye.

Visual fields (fig. 2) showed bilateral arcuate defects more dense in the left eye than in the right. A roentgenogram showed enlargement of the sella turcica with destruction of the posterior clinoids and erosion of the left anterior clinoid due to intrasellar tumor. At operation the neurosurgeon found a chromophobe adenoma that had ballooned upward both optic nerves and chiasm.

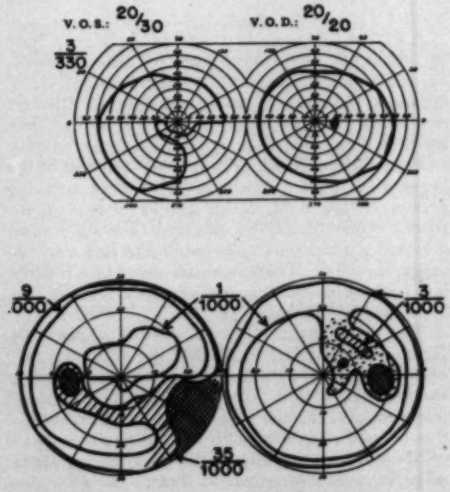

Fig. 2 (Kearns and Rucker). *Case 2.* Bilateral arcuate defects in the fields of vision.

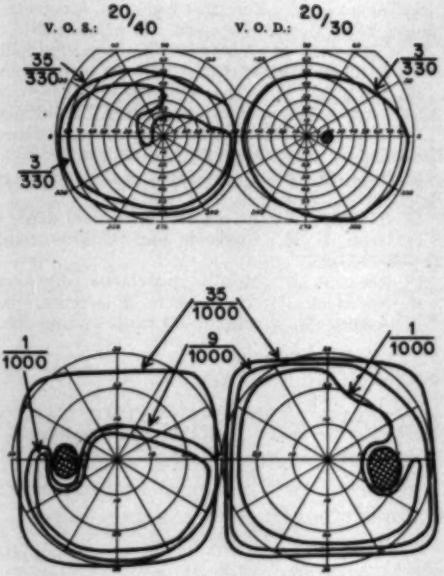

Fig. 3 (Kearns and Rucker). *Case 3.* Superior arcuate defect in the field of the left eye and a minimal superior temporal depression in the field of the right eye.

CASE 3

A 48-year-old man registered at the clinic on June 1, 1953, complaining of fatigue, loss of weight, sexual impotence, and loss of memory. He had noted some blurred vision for about six months and renewal of his driver's license had been refused because of this. The right optic disc showed minimal pallor while the left disc showed pallor, grade 1. There was no significant cupping of either disc and the intraocular pressure was well within normal limits, ranging from 15 to 25 mm. Hg (Schiøtz) in both eyes on several occasions.

The visual fields (fig. 3) revealed a dense arcuate defect located superiorly in the left eye and a minimal superior temporal depression in the right. A roentgenogram of the skull showed the sella to be enlarged, the floor depressed and the dorsum

sella, posterior clinoids, and the left anterior clinoid to be decalcified. The radiologist made a diagnosis of intrasellar neoplasm. On June 8th, the neurosurgeon performed a transfrontal craniotomy and removed a chromophobe adenoma of the pituitary.

CASE 4

A 24-year-old woman was seen at the clinic in September, 1945, because of amenorrhea of 26 months' duration and obvious signs of pituitary failure. A roentgenogram of her head showed moderate enlargement of the sella with erosion "probably caused by an intrasellar neoplasm." She had no visual complaints and the visual fields were normal on the perimeter with a 3/330 white target and on the tangent screen with a 1/1,000 white target. The optic discs were of normal color.

X-ray therapy of the pituitary was recommended and carried out. Four months later and again seven months later the visual fields were still normal. On her return visit in May, 1946, visual fields were again plotted (fig. 4); a slight but definite arcuate defect was demonstrated in the superior field of the left eye. At operation, a chromophobe adenoma was removed.

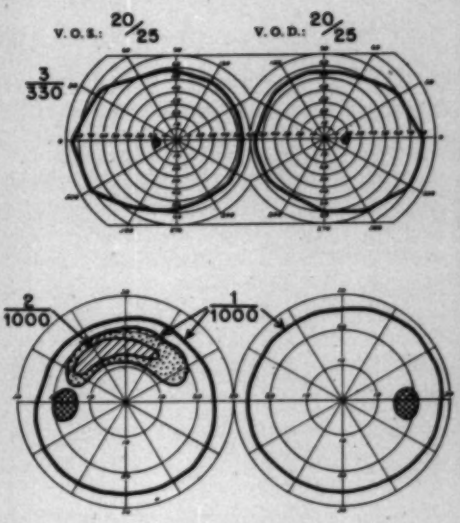

Fig. 4 (Kearns and Rucker). *Case 4.* Small arcuate defect in the field of the left eye.

COMMENT

In cases of this type, the physician is not able to make a diagnosis of chiasmal lesion from the perimetric fields alone. However, with the knowledge that such defects do occur he is able to note that pituitary tumors may produce such defects, and if there are other findings such as an enlarged sella turcica or endocrine disturbances he should arrive at a correct diagnosis and institute proper therapy.

Although we do not understand why such arcuate defects occur in these patients it is interesting to speculate on the mechanism of their production. They must certainly be due to vascular changes in the optic nerve rather than the chiasm since they cross the midline and do not have bitemporal characteristics. It is difficult to understand how direct pressure of the pituitary tumor on the chiasm alone could produce them. Perhaps even the ordinary bitemporal defects have more of a vascular basis than is usually supposed, and are not altogether due to direct mechanical pressure.

The occurrence of arcuate defects in patients with pituitary tumors is admittedly rare. However, this possibility must be kept in mind, and if the cause for an arcuate defect is not established by ophthalmoscopic examination, roentgenograms of the head should be obtained. It is not safe to assume that an arcuate defect is due to an idiopathic vascular lesion of the anterior portion of the optic nerve without first excluding the possibility of a chiasmal lesion.

SUMMARY

Chromophobe adenomas of the pituitary gland occasionally produce arcuate defects in the visual fields without bitemporal characteristics. Four cases that demonstrate this occurrence are presented. The exact mechanism by which such defects are produced by chromophobe adenomas is not clear, but the perimetrist should be aware of the possibility of such an occurrence and if the cause for an arcuate defect is not obvious, he should obtain a roentgenogram of the head.

Mayo Clinic.

COATS' DISEASE: TELANGIECTATIC OR MULTIPLE VASCULAR ORIGIN?*

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Since the first descriptions of Coats' disease, there has been controversy as to which specific vascular disease or diseases of the retina lead to the full-blown picture. The elements of this controversy have intrigued me since 1939 when my first such case was seen. When Reese[1] published his paper and the cases of telangiectasis of the retina which he had presented before the Chicago Ophthalmological Society in 1955, the difference in the retinal appearance of his cases suggested this presentation.

Although early considerations of the nature of Coats' disease were interpreted broadly, there has been a trend, started by Junius[2] and supported by Elwyn[3] and most recently by Reese,[1] to consider the disease as strictly limited to telangiectasis of the retinal vessels.

Coats,[4] himself, originally divided his cases into three groups: (1) those in which there was no gross vascular disease, (2) those with marked vascular changes, and (3) those with arteriovenous communications. He later excluded the third group as cases of angiomatosis retinae. The cause of the condition was believed by Coats to be hemorrhages into the retina from the capillaries of the external reticular layer. These were considered to be noninflammatory and to result in secondary necrosis of surrounding retinal elements and organization and encapsulation of the larger hemorrhages and transudates. Coats mentioned dilatation of small vessels in the affected retinal areas in seven of nine pathologic reports. In one of these the dilated vessels appeared like a cavernous angioma. Cases similar to this one have been reported by Miyashita and Nisyake.[5]

Leber[6] pointed to "multiple retinal aneurysms associated with retinal degeneration," a description which I believe applies to the cases to be presented in this paper.

Junius,[7] in 1934, suggested that the tiny aneurysms in the retina in Coats' disease may be equivalents of hereditary hemorrhagic telangiectasis (Osler's disease) in which there occur numerous telangiectases on various parts of the body, as part of a familial and hereditary disease. Although a few reported cases suggest simultaneous telangiectatic involvement of the skin, no hereditary aspects were found by Reese or in the cases herein reported.

Elwyn,[3] after studying the cases reported by Coats and others, presented a comprehensive review of Coats' disease and pointed out that the fundamental pathologic element is a congenital vascular malformation corresponding to telangiectasis and involving the small vessels with the formation of miliary aneurysms and capillary and venous dilatation with defective vessel walls. He considered the exudates and fibrovascular formations in the retina to be the result of local circulatory disturbances following transudation of plasma and blood.

Reese[1] supported the telangiectasis basis of Coats' disease with excellent drawings and cases, two of which were followed through the stages from telangiectasia of the retina to full-blown Coats' disease. He agreed with Elwyn that the retinal vascular lesion should be viewed as telangiectasis rather than cavernous hemangioma since the latter have a stroma of their own while the telangiectases have retinal tissue as their stroma.

Virchow[8] had classified the racemose angiomas as having a stroma of tissue other than their own. They were divided into four subgroups: (a) telangiectasia in which the dilated vessels are probably capillaries, (b)

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angioma racemosum arteriole, (c) angioma racemosum venosum, and (d) aneurysma arteriovenosum, in which there is a direct connection between the dilated arteries and veins.

Aside from the angiomatous lesions considered above, small miliary lesions described as aneurysms have been reported but are considered to be rare (Duke-Elder[9]). They were described in healthy young persons but were more typical of age and atheromatous vascular degeneration.

Reese pointed out that, although telangiectasia of the retina should be viewed as a precursor of Coats' disease, the progression to the latter disease should not be considered obligatory since such telangiectasis may remain static, give rise to intermittent hemorrhages with no serious secondary changes, or go on to the complete picture of Coats' disease where there is retinal detachment of the characteristic type.

Clinically, Coats' disease occurs mostly in young persons and affects usually one eye, although bilateral involvement does occur. The visual loss is due to retinal exudate which is single or multiple and flat or prominent and is located anywhere in the fundus. The exudate is white or yellowish white, grayish or greenish in color and lies mainly behind the retinal vessels. Hemorrhage is usually present. Glistening cholesterol deposits are frequent. Pigment deposits are occasionally found.

Case reports

In the cases which I have seen, only one was followed from a fairly early stage to the stage of Coats' disease. In four, the condition was of the type seen in children and in six others the same type of globular vascular lesion was seen as a degenerative process in adults of middle or old age. All showed lesions differing from those described by Reese, although several showed telangiectatic vessels in addition. I believe many of these latter to be secondary.

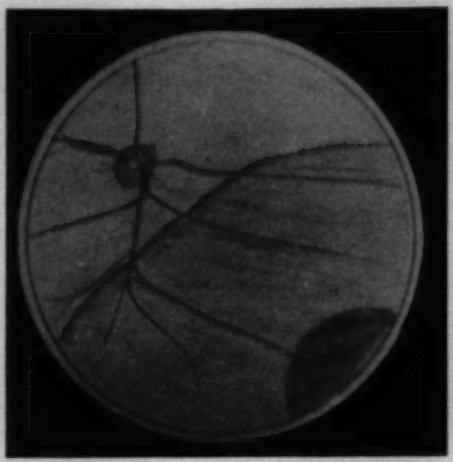

Fig. 1 (Sugar). *Case 1.* Sketch to show area of retina involved by exudate and the moundlike lesion in the lower retinal periphery.

Case 1

This 21-year-old soldier was seen by me on January 8, 1945. He gave a history of sudden sharp pain in the right eye with associated decrease in visual acuity one month previously. This episode was followed by a similar episode one week later. There was no previous history of ocular disease. No family history of ocular difficulty was elicited. The visual acuity was 20/70 corrected, R.E., and 20/15, L.E. The lower-half of the right fundus was diffusely yellow, demarcated sharply from the normal retina above. The retina in the involved area contained many crystals and some telangiectatic vessels. Down and nasally in the extreme periphery there was a moundlike red lesion which could not be studied more exactly (fig. 1). The tension was normal. A diagnosis of Coats' disease was made. The vision of the right eye decreased gradually until on February 20, 1945, it was 20/100 corrected (+1.0D. sph.). Some hemorrhages were visible in the periphery of the retina in the region of the mass noted previously.

On March 17, 1945, under local anesthesia, the conjunctiva was incised 10 mm. from the limbus in the lower nasal quadrant and the sclera bared. Partial penetrating diathermy with a Gradle needle was applied between the medial and inferior recti in an area four to 10 mm. from the limbus. The incision was closed. After healing, the patient was discharged and not seen until May 15, 1946. He stated that there had been no visual loss until one and one-half weeks previously when it suddenly became poorer. The visual acuity was reduced to light perception in the lower field. The entire retina was yellow and many cholesterol crystals were visible. On January 10, 1947, the patient gave a

Fig. 2 (Sugar). *Case 2.* Aneurysm in retinal periphery. This was bright red in the original color photograph.

history of loss of light perception about September 1, 1946. There was no pupillary reaction to light. The retina was elevated in places. The tension was normal.

This case was considered to be one of Coats' disease and was followed for two years beginning shortly after its onset. An angiomalike lesion was barely visible and was treated by scleral diathermy unsuccessfully. This may have been due to the size of the lesion or to its inaccessibility to view, or both.

CASE 2

This 14-year-old Negro boy was first seen by me in October, 1946. His right eye was entirely normal. The left visual acuity was 15/200. It was thought that the acuity had been poor since a blow to the eye three weeks previously. Along the superior temporal vessels were a number of small aneurysms, some connected with neighboring vessels (fig. 2). The area above the disc showed feathery exudate along the vessel walls and deep to the vessels. Temporal to the edematous macular area was a large area of white exudate which appeared vaguely to suggest the choroidal vascular pattern (fig. 3). At the lower edge of this area there appeared to be a slate-gray area, suggesting an elevation of the retinal pigment epithelium. Folds of epithelium and retina extended toward the disc.

Physical examination was otherwise entirely normal. Neurologic examination, including skull roentgenograms and lumbar puncture, were negative. Serologic examinations for syphilis were negative.

When last seen at the University of Illinois Research Hospital in May, 1949, the left vision was reduced to finger counting. The exudate had extended through the macula, with the addition of a star-shaped figure.

The fundus picture in this case is particularly interesting because of the differing appearance of the exudate in different portions of the fundus and is the best means of explaining the general fundus picture. The large white area suggests that in this area the pigment epithelium was obscured by the exudate. My early impression was that the pattern was that of the choroidal vessels but I am now convinced that this is simply the peculiar nature of the retinal exudate itself. In the gray area just below the white area the pigment layer appears to be elevated. The folds result from this elevation. The nebulous exudate above the disc and surrounding the retinal vessels appears to be exudate deposited in the retina by phagocytic cells. The phagocytes are probably the ghost cells of Coats' which, according to Leber are derived from the pigment epithelial cells. These cells not only phagocytose but probably also form connective tissue which later tends to localize the areas of involvement and, still later, lead to contracture and retinal separation. The migration to the vessel walls and, in localized cases, the later complete or partial disappearance of the perivascular exudate is evidence of the process of phagocytic deposit and transport. The phagocytic function of the pigment epithelial cells fits the ideas of Krückmann[10] but not those of Wolter, Goldsmith, and Phillips,[11] who consider the macrophages to be retinal microglial cells. I see no reason why both types of cells might not be involved.

CASE 3

This 12-year-old girl was seen by me in 1946. Her right eye was entirely normal. The left visual acuity was reduced to 20/200 due to a macular star figure (fig. 4). Temporal to the

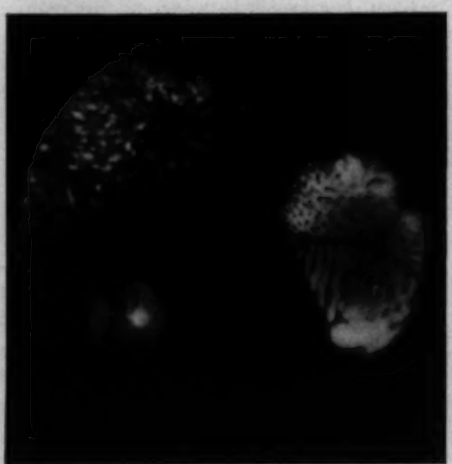

Fig. 3 (Sugar). *Case 2.* Fundus photograph of exudate in retina in boy, aged 14 years.

Fig. 4 (Sugar). *Case 3.* Fundus photograph to show the macular star figure and exudate temporal to the macula, close to the area shown in Figure 5.

macula two round angiomalike lesions were visible, with surrounding exudate (fig. 5).

CASE 4

This patient was aged 36 years when seen by me in 1939. She had been seen at the University of Iowa Clinic in 1935 because of pain in the right side of her head and loss of vision in her right eye. The right retina was detached and seemed to be filled with new vessels. The left fundus showed

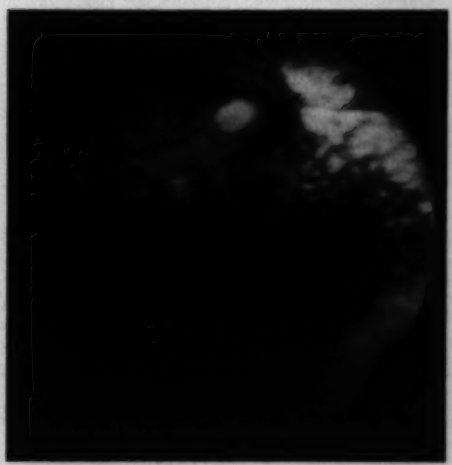

Fig. 5 (Sugar). *Case 3.* One of the two angiomalike lesions in equatorial region surrounded by exudate in retina.

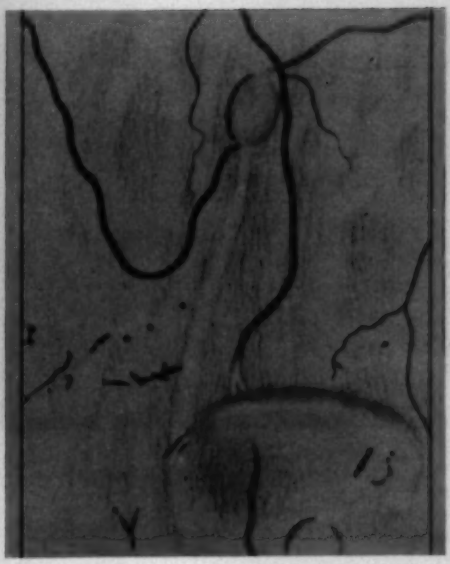

Fig. 6 (Sugar). *Case 4.* Drawing made in 1935 at the University of Iowa Clinic, showing oval lesions in lower left fundus. (Courtesy University of Iowa Clinic.)

an oval lesion one-third disc diameter in size; in the course of one of the peripheral veins another such lesion unrelated to vessels was visible peripheral to the first one (fig. 6). A diagnosis of Coats' disease was made.

Examination in 1939 showed no light perception in the right eye and 20/70 vision, corrected, with the left. The right eye had a dense cataract and a tension of 82 mm. Hg (Schiøtz). The left eye showed telangiectases and varicosities of small vessels. One inferior temporal vein was very tortuous and led to a raised angiomalike mass (fig. 7). What appeared to be an artery leading to the mass was found to be unrelated to it and could be identified in the drawing made in 1935.

The retina was raised below by considerable exudate. Neurologic and X-ray studies showed no evidence of intracranial angiomas.

The right eye was enucleated. The globe showed degenerative changes resulting from glaucoma, anterior subcapsular cataract, and marked retinal degeneration. The retina was completely separated (fig. 8). The normal structure was unrecognizable and consisted of glial and connective tissue with cystic spaces and some large vessels with hyalinized walls.

The left eye was treated with 4.30 mc. radon in two gold implants on July 25, 1939. When last heard from in March, 1943, there had been no further visual deterioration, according to the patient.

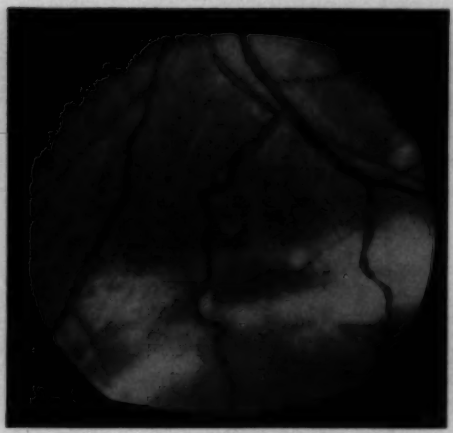

Fig. 7 (Sugar). *Case 4.* Fundus photograph, left eye, to show appearance in 1939. One angiomatous mass was present just below the lower edge of photograph. The extent of the exudate is shown.

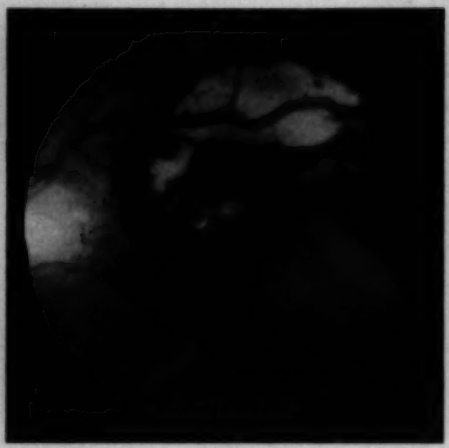

Fig. 9 (Sugar). *Case 5.* Fundus photograph to show preretinal hemorrhage below area of aneurysmlike mass. Deep retinal exudate is present above.

This case is important in indicating further that bilateral involvement may be encountered. The probability that the use of radon was successful in preventing further deterioration must be considered.

These first four cases were the only ones which occurred in young people. The lesions in all suggested aneurysmal lesions but this picture could also be produced by telangiectasia. However, they do show a somewhat different picture from the cases described by Reese.

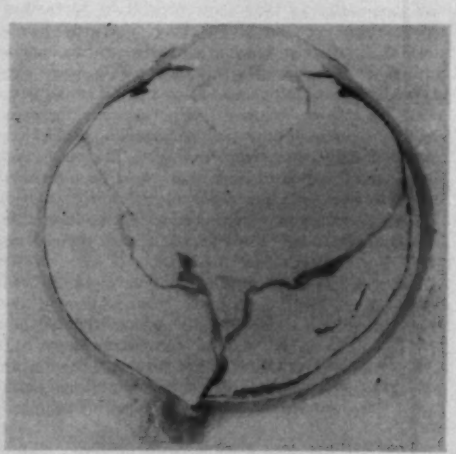

Fig. 8 (Sugar). *Case 4.* Low-power photograph of section of right eye to show vascularized thick areas of separated retina. (Slide courtesy Dr. Georgiana Theobald.)

CASE 5

This 68-year-old hypertensive man was seen on September 25, 1956, because of gradual loss of vision in the left eye during a period of one and one-half months. The vision in this eye was reduced to finger counting. A round preretinal hemorrhage was present in the macular area and there was an area of telangiectasia and deep retinal deposition above this (fig. 9). When seen in March, 1957, the hemorrhage had disappeared, leaving some pigmentary macular change and a round connective tissue mass in the area of the "leak" (fig. 10).

This case indicates the potentiality for resorption of exudate in the same manner that hemorrhage is cleared.

CASE 6

This 75-year-old woman was seen on October 10, 1956. She had had mild diabetes for 10 years and hypertension for several years. The left visual acuity was reduced to 20/70 corrected. A group of aneurysmlike lesions were present above the left macula. A circinate type of deposit was present around this (fig. 11). No other evidence of diabetic retinopathy was present in either eye. The deposits had increased in extent by the last examination six months later (fig. 12).

CASE 7

This 64-year-old hypertensive man was first seen on November 12, 1955. His left eye had been

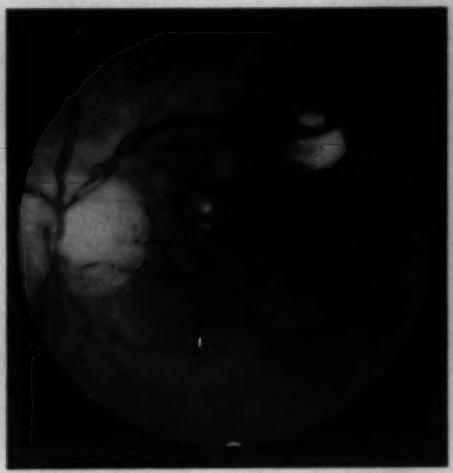

Fig. 10 (Sugar). *Case 5.* Same eye as Figure 9, six months later.

injured with a piece of steel 39 years previously. The left visual acuity was reduced to 20/400 corrected. A macular hemorrhage with exudate above and below was present. Exudate of the type which appeared to lie behind and to ensheath the vessels was present in the vicinity of the superior temporal vessels just above the macula (fig. 13). When last seen in March, 1957, the macular hemorrhage was gone (fig. 14) but the exudate involved the entire macular area (fig. 15).

CASE 8

This 74-year-old man was first seen by me on April 27, 1948, for routine refraction. He had had a pituitary tumor removed seven years previously. His visual acuity, fundi, and tension were entirely

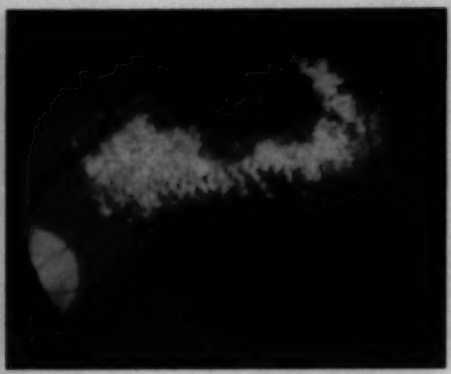

Fig. 12 (Sugar). *Case 6.* Same eye as in Figure 11, six months later. Note increase in circinate deposits.

normal. He was re-examined in September, 1951, and needed a change in glasses because of decreasing hyperopia. The findings were otherwise normal. In December, 1952, on examination, exudation was found along the upper portion of the left optic nerve with what appeared to be a vascular aneurysmlike anomaly just next to the disc (fig. 16). The vision remained normal until the middle of January, 1954, when the patient suddenly noted decreased visual acuity with the left eye. On examination, the left visual acuity was reduced to finger counting because of extension of the exudative process to the macula (fig. 17). When last seen on March 5, 1957, the exudate had extended in all directions and contained many areas of cholesterol crystals (fig. 18).

This case is important in indicating the possibility of the acquired nature of the lesion, particularly since he had been examined twice previously over a period of four years. Although it is true that a

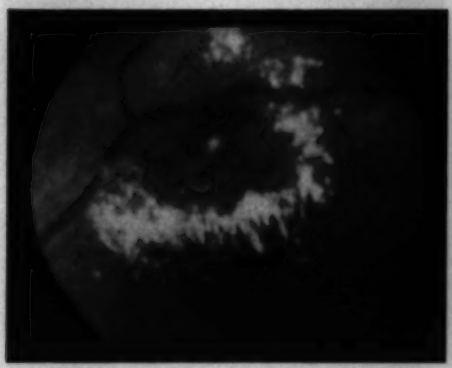

Fig. 11 (Sugar). *Case 6.* Fundus photograph to show aneurysmlike lesion and circinate retinopathy.

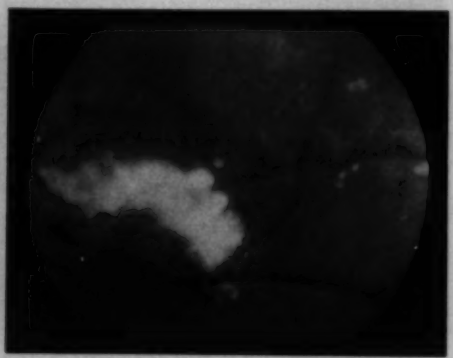

Fig. 13 (Sugar). *Case 7.* Exudate surrounding aneurysmlike lesions in vicinity of superior temporal vessels.

Fig. 14 (Sugar). *Case 7.* Same eye as in Figure 13, 16 months later. The involved superior temporal area was "healed" but a macular figure was present (fig. 15).

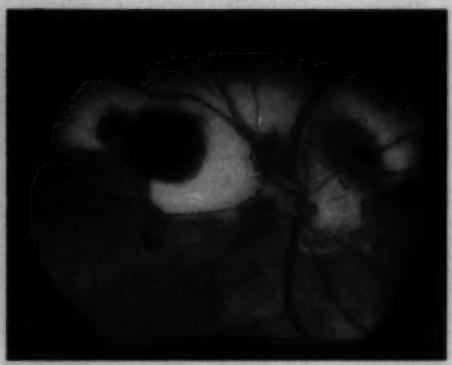

Fig. 16 (Sugar). *Case 8.* Fundus photograph (December, 1952) of 74-year-old man with aneurysmlike lesions (dark areas) surrounded by deep retinal exudate.

small congenital vascular lesion might be overlooked, it is equally reasonable that in a 74-year-old man a degenerative, completely acquired cause might be expected.

CASE 9

This 72-year-old physician was first seen by me on April 24, 1953, because of blurring of vision in the left eye which had begun with pain two weeks previously. The visual acuity in this eye was reduced to 20/40 with correction because of some vitreous hemorrhage. Multiple small aneurysmal dilatations of the retinal vessels with circinate deposits were present (fig. 19). Some feathery perivascular exudate was present. There was no familial history of telangiectasis. When last examined in March, 1957, there had been regression of the exudate in the main area of involvement (fig. 20).

CASE 10

This 47-year-old woman was first seen in December, 1951, because of momentary sharp pain in the right eye. The visual acuity was normal. The only abnormality was a loss of pigment epithelium around the upper portion of the right disc with a large choroidal vessel visible. A small aneurysm-like lesion was present in the center of this area

Fig. 15 (Sugar), *Case 7.* Macular exudate in same eye as Figure 14.

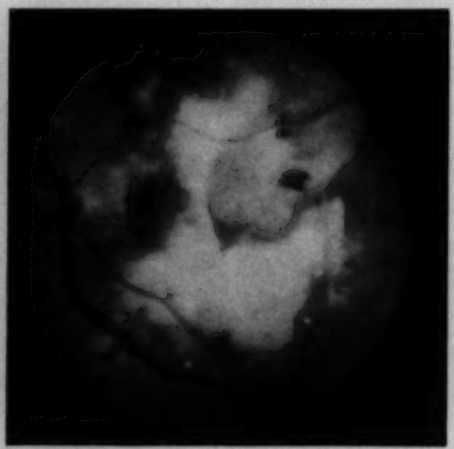

Fig. 17 (Sugar). *Case 8.* Same eye as Figure 16, two years later, showing marked increase in exudate.

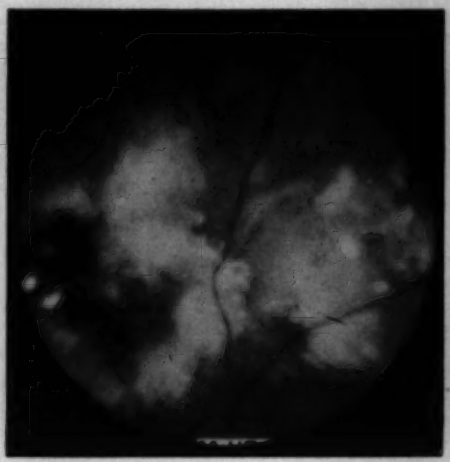

Fig. 18 (Sugar). *Case 8.* Same eye as Figure 16, five years after onset, to show exudate containing cholesterol crystals (sharp white).

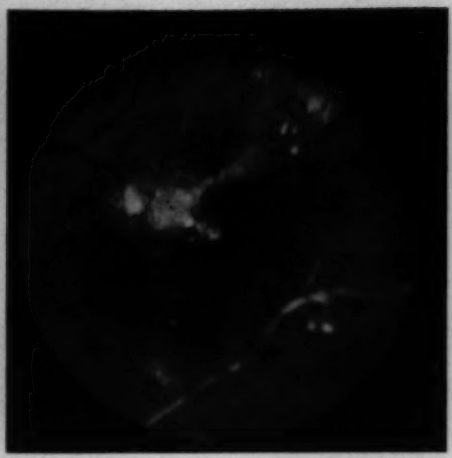

Fig. 20 (Sugar). *Case 9.* Same eye as in Figure 19, four years later, to show marked regression of exudate.

(fig. 21). No inflammatory activity was visible. When seen again on February 12, 1953, the right vision was reduced to 20/200 because of hemorrhage and extension of the previously noted process with an angiomalike lesion at the temporal extremity of the lesion (fig. 22). When last seen on April 19, 1956, there was a hemorrhage surrounding the macular lesion (fig. 23).

The interpretation of this case as one of Coats' disease and even its inclusion in this group might be criticized. It may be related to Kuhnt-Junius disease but is atypical in having apparently two foci adjacent to the disc. The slow progression may be more related to the slight degree and intermittency of leak. The condition might also be considered to be a form of choroiditis except that there was never that type of pigmentary reaction during healing nor was there ever any suggestion of any exudate other than hemorrhage.

Fig. 19 (Sugar). *Case 9.* Dark areas of aneurysmal-like nature surrounded by deposits of the circinate type.

Case 11

This 93-year-old woman was followed by me for nearly 20 years. Five years ago she first began to develop senile macular degeneration in the left eye and slight pigmentary changes in the right macula. In 1956, her vision was reduced to finger counting. A typical ring of circinate type encircled the right macula. In the center of the ring the retina appeared slightly grayish and elevated, as in Kuhnt-Junius disease. However, a small aneurysm-like lesion was present in the foveal area.

Comment

The 11 cases described herein all showed

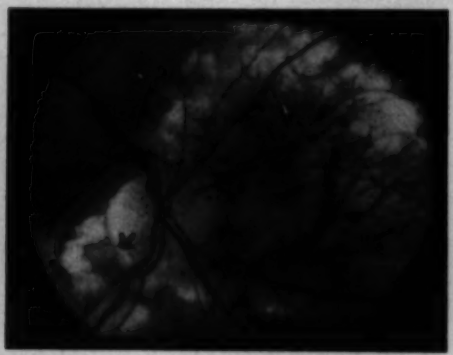

Fig. 21 (Sugar). *Case 10.* Fundus photograph to show one of aneurysmlike lesions temporal to the right disc (arrow), and depigmentation of recent origin.

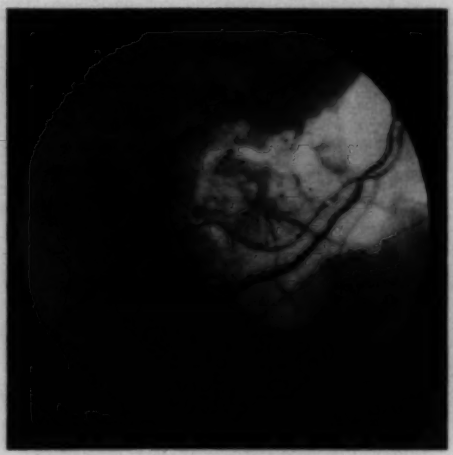

Fig. 22 (Sugar). *Case 10.* Same eye two years later, to show extension of temporal area. An angiomalike lesion is present at its temporal extremity.

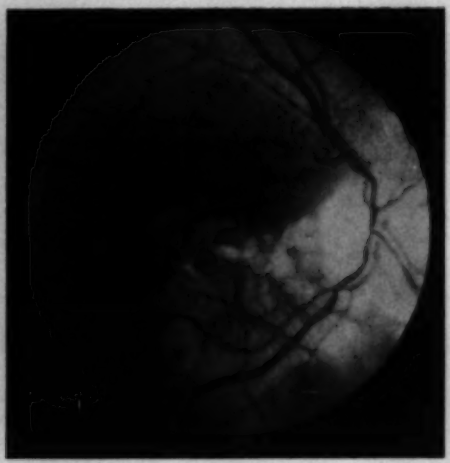

Fig. 23 (Sugar). *Case 10.* Same eye three years after Figure 22, following a quiescent period. Fresh hemorrhage is seen around the vascular lesion.

lesions differing in appearance from those shown in the drawings in Reese's paper. However, several showed telangiectatic vessels in addition to the lesions which I consider characteristic. I believe that at least some of the telangiectases are secondary. Reese described the characteristic fundus lesion leading to Coats' disease as "a circumscribed slightly elevated area over which are numerous small sharply outlined red globules." The lesions described in this paper are similar to those described by Leber. The latter were considered by Reese to be of the same nature as those he described.

I believe that the primary pathologic process may be telangiectatic or aneurysmal or even of other nature, including von Hippel's disease. This is, indeed, a regression in our thinking to Coats' original description but I believe it to be accurate. It is the exudate and its deep location in the area of nutrition by the choroid which is the common denominator in these cases. The degree of involvement is undoubtedly related to the degree of "leak" from the vascular lesion. If it is very slight, it is understandable that diffuse exudation will not occur, as in Case 10, while continued, more profuse leak will involve the entire retina. It is probably true that the lesion might even "heal" by thrombosis within the vessel or sealing off by connective tissue around the vessel. This certainly can occur in older individuals as in Cases 5, 9, and 11.

The type of exudate seen in Case 2, as well as in others, makes one speculate as to its nature and location. Since the main exudate in these cases lies behind the retinal vessels, it must lie between the retina and pigment pithelium. At first the exudate is loose and cellular and later becomes infiltrated by connective tissue cells. Calcium deposits occur here and there. My impression is that the exudate is largely serum and blood derivatives which become phagocytosed in part, in some cases even causing the pigment epithelial cells to take on the function of phagocytes which transport some of the exudate to deposit it around vessel walls where it is seen as irregular sheathing. This location of the exudate was not shown in Reese's drawings. I consider it characteristic, but it does not occur in all cases.

All cases have in common a noninflammatory exudative retinopathy in the posterior layers of the retina which is the original

description of Coats' disease. Since it is almost impossible in each case to determine the exact nature of the vascular lesion clinically it is perhaps best to designate them as juvenile or adult types, signifying, thereby, simply that the adult type may be more often of the acquired miliary aneurysm type while perhaps the juvenile type may have a congenital telangiectatic origin. All involve lesions which are derived from the capillaries in the deepest retinal layers where nutrition is normally derived from the choroid. It is possible that some of them result from an attempt to compensate for a choroidal vascular insufficiency or local retinal venous occlusion.

Summary

Coats' disease is held to be the result of vascular "leak" in eyes with telangiectasia, miliary aneurysms, or other deep retinal vascular lesions and is not a specific entity, etiologically. The exudate is considered to be derived largely from blood serum in some cases while in others it probably comes also from degenerated retinal tissue elements. Some of it is phagocytosed and transferred to ensheath the retinal vascular walls while some remains localized and may become organized; and some may appear as circinate or star figures.

18140 San Juan (21).

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Ophthalmic Miniature

"When I had given up inquiring into real evidence," he (Socrates) proceeded, "I thought that I must take care that I did not suffer as people do who look at the sun during eclipse. For they are apt to lose their eyesight, unless they look at the sun's reflection in water or some such medium. That danger occurred to me. I was afraid that my soul might be completely blinded if I looked at things with my eyes, and tried to grasp them with my senses."

Plato, *Phaedo.*

HISTOPATHOLOGY OF PRIMARY ENDOTHELIAL-EPITHELIAL DYSTROPHY OF THE CORNEA*

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New York

In 1910, Fuchs first described the full clinical picture of what he called "dystrophia epithelialis corneae."[1] This was prior to the era of slitlamp biomicroscopy. Until Gullstrand's introduction of the slitlamp into the ophthalmologic field in 1911, the corneal endothelium could not be demonstrated clinically and the importance of the endothelium in the development of this condition was not known.

Earlier writers, Koeppe in 1916[2] and Kraupa in 1920,[3] had suggested an endothelial defect in eyes with epithelial dystrophy, but the first clinical description of the nature of these endothelial changes was made by Vogt in 1921,[4] when he introduced the new technique of specular reflection in slitlamp biomicroscopy. He described the condition as "droplike endothelial prominences." In 1930, in his *Atlas of Slitlamp Microscopy of the Living Eye*, he described it as "cornea guttata."[5]

Vogt's findings opened the road to further clarification of the picture. Most observers (Vogt, 1921, Graves, 1924,[6] Friedenwald and Friedenwald, 1925,[7] Goar, 1934,[8] Lloyd, 1944,[9] Stocker, 1953,[10] Thomas, 1955,[11] and Frayer, 1956[12]) recognized that the so-called Fuchs', or Fuchs' epithelial, dystrophy represents a late stage of the much more common condition of endothelial dystrophy, cornea guttata. The Friedenwalds[7] emphasized this fact. Stocker,[10] out of his series of 25 cases, observed the development of endothelial-epithelial corneal dystrophy in six cases which had previously exhibited only endothelial changes.

Owing to the relatively infrequent occurrence of the late manifestation represented by the type of epithelial dystrophy described by Fuchs, endothelial dystrophy and epithelial dystrophy are still listed and sometimes reported as separate entities. This fact complicates and confuses a study of the literature on this subject. The term "primary endothelial-epithelial corneal dystrophy" is, in our opinion, more accurately descriptive of this disease.

Although a large number of clinical observations of endothelial-epithelial dystrophy of the cornea have been reported, a review of the literature reveals surprisingly few histologic studies. Up to the time of Frayer's[12] paper in 1956, the histologic examination of only eight eyes with this type of dystrophy had been reported. Since then three additional cases have been reported by Irvine.[13] The first few histologic reports of this disease were made by Vogt in 1930, followed by von Hippel (1932),[14] and Goar (1934). In 1944, Lloyd reported histologic studies by Verhoeff; Calhoun,[15] in 1951, and Stocker, in 1952[16] and 1953, reported further cases. Throughout the literature the histologic findings are described as degenerative changes in the corneal endothelium with nodular excrescences on Descemet's membrane and occasional duplication of Descemet's membrane.

In Stocker's[10] unique flat preparation of corneal endothelium in cornea guttata the characteristic degeneration of the endothelial cells is well demonstrated but the nodular excrescences on Descemet's membrane are still not clearly seen. As he states, "the light area in the right upper corner probably represents a wart without endothelial covering."

A search of the literature failed to reveal any report of phase-contrast microscopic examination of a flat preparation of corneal endothelium in this type of dystrophy. In our own studies using this technique, we

* From the laboratory of The Eye-Bank for Sight Restoration, Inc. This study was aided by Grants B-1130 and B-153 from the National Institutes of Health, U. S. Public Health Service.

found that both the endothelial changes and the nodu'ar excrescences on Descemet's membrane were amazingly clear. Phase-contrast microscopy was much superior to ordinary light microscopy, especially in showing the early stages of the excrescences on Descemet's membrane.

The purpose of this paper is to present additional observations on the histopathology of primary endothelial-epithelial dystrophy of the cornea, to discuss the mechanism leading to the development of the pathologic changes, and to correlate these changes with the clinical findings.

Material and methods

Our material consisted of pathologic discs removed at keratoplasty. A total of 28 cases with a clinical diagnosis of Fuchs' dystrophy or corneal dystrophy were studied. The material* was collected from the Manhattan Eye, Ear, and Throat Hospital, and covers a period from February, 1952, through April, 1957. Each disc was fixed immediately in 10-percent neutral formol-saline solution, except for four which were fixed after supravital staining with alzarin red to demonstrate the intercellular cement substance of the endothelium. The fixed tissue was bisected and half of the disc was sectioned serially in the transverse plane. The posterior portion of the other half was used to make a flat preparation and the anterior part for flat serial sections.

The flat preparation, involving the endothelium, Descemet's membrane, and as little as possible of the stroma, was stained with hematoxylin only and mounted to study the endothelium. After the flat preparation was studied with the ordinary light microscope as well as the phase-contrast microscope, it was embedded in paraffin for thin, flat serial sections.

Paraffin or double-embedded serial sections were stained variously with hematoxylin and eosin, Verhoeff's elastic tissue method, toluidine blue, van Gieson and periodic acid-Schiff stain. They were prepared for both quantitative and qualitative correlation of the pathologic process found. In 20 of the 28 discs the pathologic diagnosis was endothelial-epithelial corneal dystrophy. In this series six discs were from males, 14 from females; the youngest patient was 42 years of age.

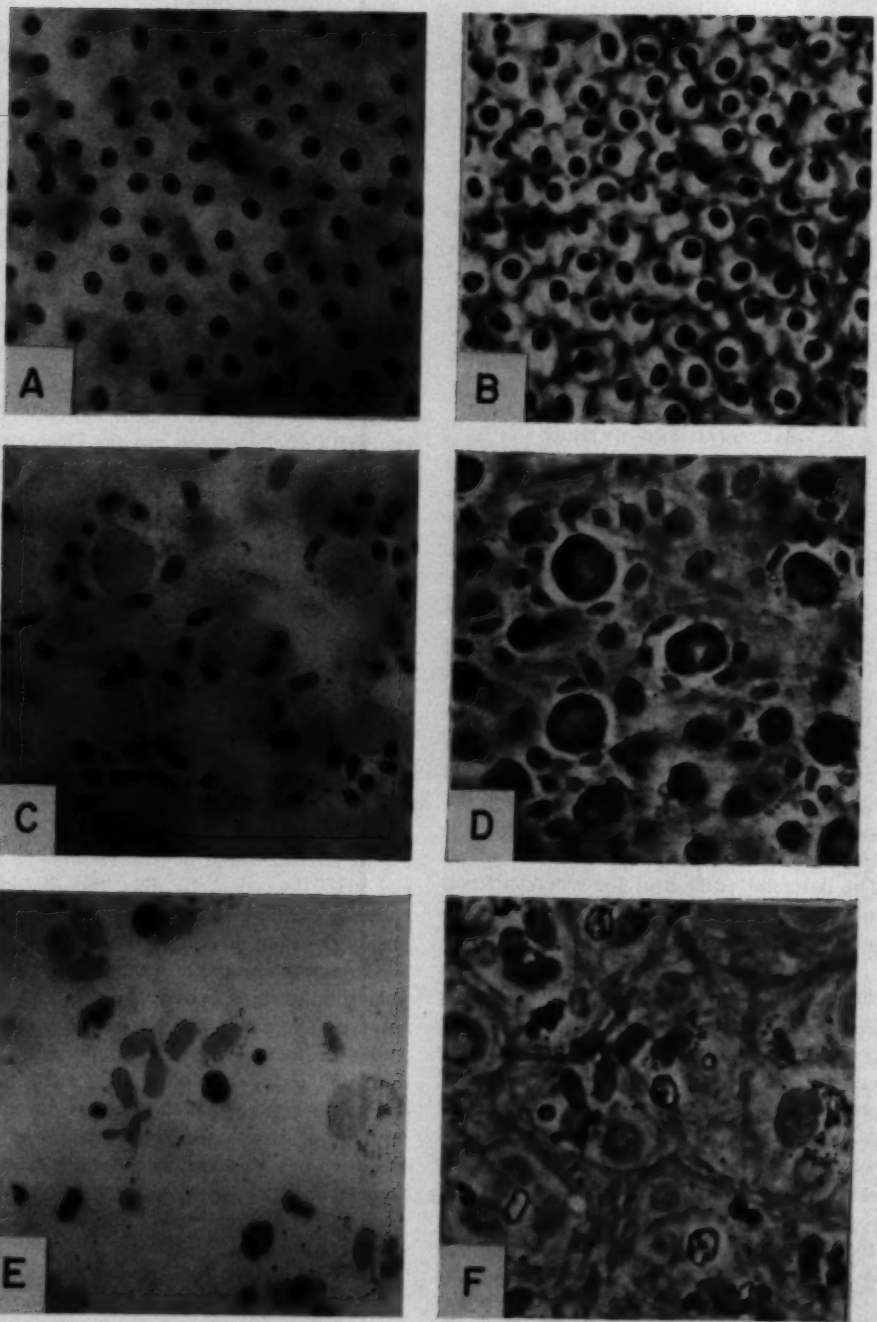
* We are indebted to Dr. R. Townley Paton for supplying most of the material that made this study possible.

Histologic findings

Endothelium and Descemet's membrane

Flat preparation. The most characteristic histologic findings of endothelial-epithelial dystrophy of the cornea are the Descemet's membrane and endothelial changes. The nuclei, which are normally round or oval and evenly distributed (figs. 1-A and B and 2-A and B), are distorted by globular bodies of different sizes (fig. 1-C and D). The displaced, crowded nuclei are of various sizes and shapes and exhibit pyknotic changes (fig. 1-C and D). They appear round, oval, rod, dumb-bell, or sickle-shaped. Some of them seem to have been pushed aside by the globular bodies (fig. 1-C and D). This seems especially true of the cells whose nuclei are dumb-bell or sickle-shaped (fig. 2-C and D).

The normal round or oval endothelial nuclei stain less densely than the distorted, pathologic nuclei (fig. 1-C). The globular bodies show up strikingly under phase contrast microscopy (fig. 1-D), and small globular bodies in the early stage of development can be seen, which are not apparent under ordinary light microscopy (fig. 3-A and B). Using the phase contrast microscope, these small globular bodies appear to be intracellular, cytoplasmic bodies of low density (figs. 4-B and 5). As they become larger, they show a denser margin surrounded by a clear halo (fig. 3-B). They eventually break the cell border, and fuse (fig. 6-A and B), destroying the regular pattern of normal endothelial cells (fig. 4-A). Fine pigmented granules and clumps


A
B
C
D
E
F

of pigment are scattered over the endothelium in most cases (figs. 1-C, D, E, and F; 2-E and F; 3-A and B; 4).

In four cases, and in some areas of three other cases, there were numerous globular bodies with less density in proportion to their size. They appeared to be covered by a layer of substance which was lined by cells with fairly large, faintly stained nuclei (figs. 1-E and F and 2-E and F). When these globular bodies are focused at deeper levels, they show up more distinctly.

*Cross and flat sections.** The globular bodies described above are seen as nodular excrescences on Descemet's membrane in cross sections (figs. 7, 8, 9, 10, 12, and 13). In some cases the cytoplasm of the endothelium is thinned out over the surface of the excrescences (fig. 7). In other places the endothelial cytoplasm is completely absent and the nuclei are pushed toward the valleys between the excrescences (fig. 8).

Some of the excrescences are flattened, with broad bases; some are hemispherical, ovoid, saucerlike, or anvil-shaped (figs. 7, 8, and 9). These excrescences are usually directed posteriorly when they coalesce with Descemet's membrane (figs. 7, 8, and 9). In serial section study, some globular bodies appear to be separated from Descemet's membrane, and show their biconvexity (fig. 10).

In thin flat serial sections, these globular bodies appear intracellularly as cytoplasmic inclusions (fig. 11). With a variety of tissue stains the excrescences have staining properties identical with those of the inner layer of Descemet's membrane.

A rarer finding is an additional laminated membrane overlying the posterior surface of the original Descemet's membrane with excrescences between the two layers (fig. 12). The new membrane exhibits different staining properties from the original Descemet's membrane. It stains lightly with hematoxylin and eosin (fig. 12), periodic acid-Schiff, and Verhoeff's stains. The new membrane is covered, for the most part, with endothelium, which has abnormally large, thin, elongated nuclei, irregularly grouped (figs. 1-E and F, 2-E and F, and 12). Pigmented granules may be found on the surface of the excrescenses on Descemet's membrane, within the endothelial cells themselves, or in between the two membranes (figs. 11 and 13).

Stroma. In a few cases the stroma is normal, but in most cases the corneal lamellae are infiltrated by fluid and separated into fine, light fibrillae. Loss of metachromasia in toluidine blue stain was noted in certain areas of some cases (fig. 14). There was no vascularization of the stroma.

Epithelium, basement membrane, and Bowman's membrane. The degeneration of the corneal endothelium eventually destroys the integrity of the aqueous barrier, and fluid enters the corneal stroma, causing changes in the epithelium. In relatively early stages of epithelial change, there is intracel-

* It is difficult to obtain flat sections of flat preparations. There is usually some degree of obliquity.

Fig. 1 (Chi, Teng, and Katzin). Flat preparations of normal endothelium and endothelium exhibiting changes found in endothelial-epithelial dystrophy. (A, C, and E) were taken under light microscopy; (B, D, and F) under phase-contrast. Magnification is the same in each picture (×160). (Hematoxylin stain.)

(A and B) Normal round or oval endothelial nuclei, evenly distributed (BP158).

(C and D) Endothelial nuclei of various sizes and shapes, with pyknotic changes. They are displaced by globular bodies which show up strikingly in (D) the phase contrast picture (BP120).

(E and F) In the phase contrast picture (F) the globular bodies appear less dense in proportion to their size. In this preparation they are seen through a layer of substance and regenerated endothelial cells. Pigmented granules and clumps of pigment show up very well. (E) Taken under light microscopy shows the abnormally large nuclei of these cells, which stain only faintly (BP159).

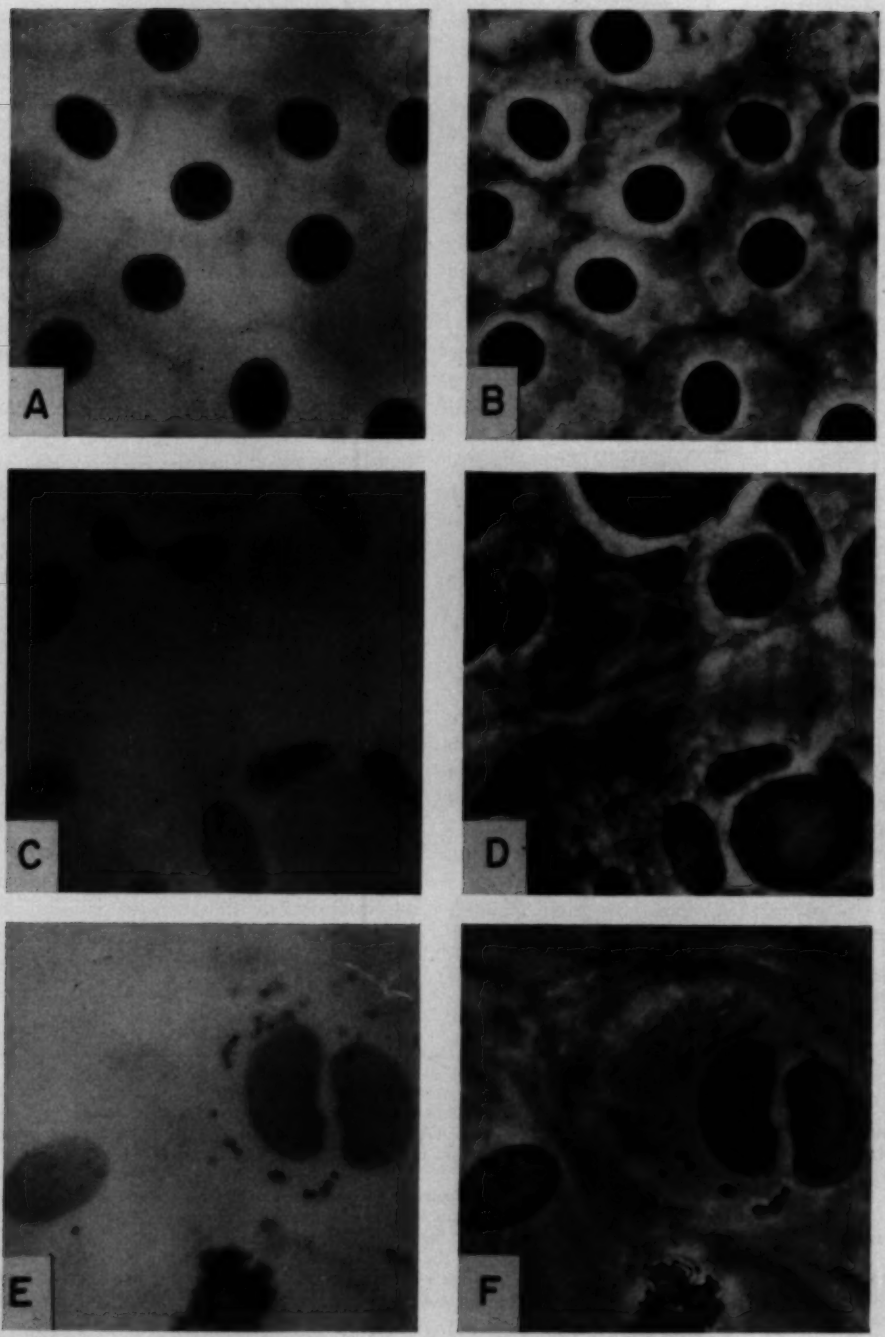


Fig. 2 (Chi, Teng, and Katzin). Higher magnification (×500) of flat preparations, of normal endothelium and endothelium exhibiting the changes found in endothelial-epithelial dystrophy. (A, C, and E)

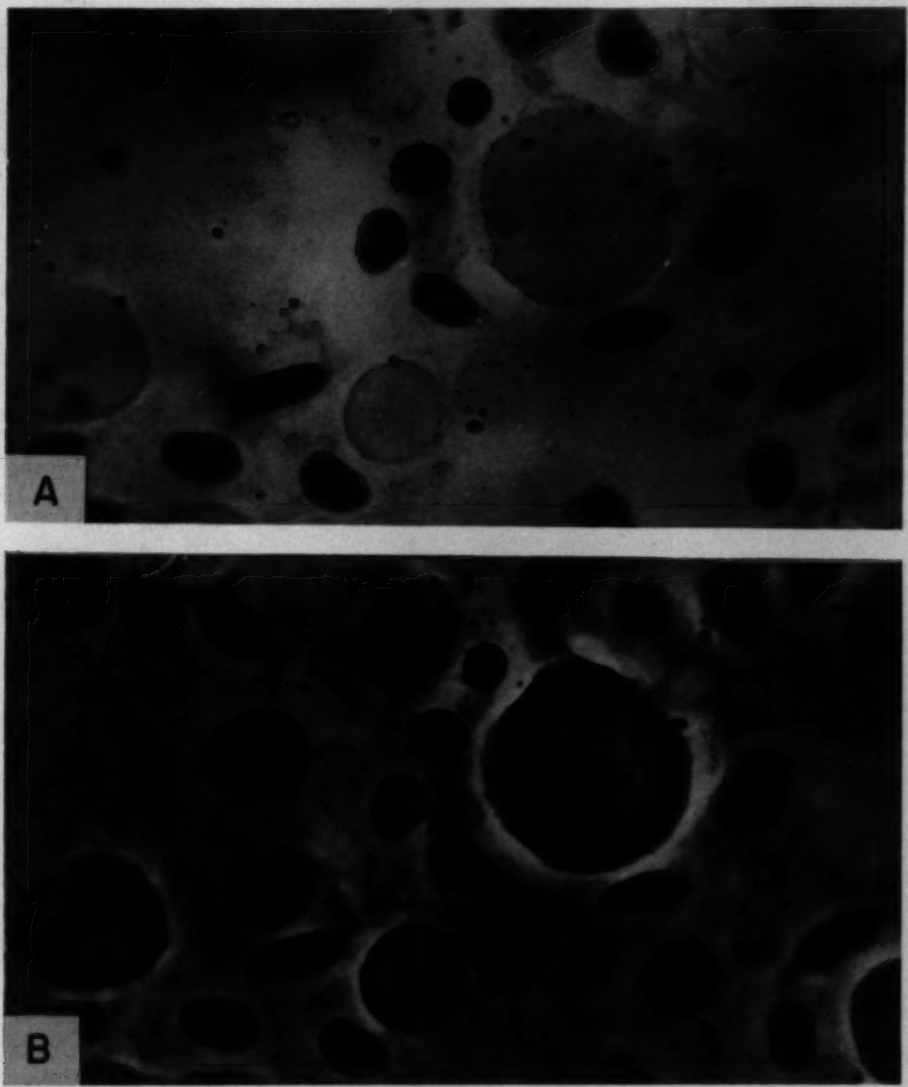


Fig. 3 (Chi, Teng, and Katzin). The small globular bodies indicated by the arrows in (B), the phase-contrast picture, are in early stages of development. The larger globular bodies have a dense margin surrounded by a clear halo. This resembles the slitlamp, biomicroscopy picture. These phenomena are not apparent under ordinary light microscopy, in (A). (Flat preparation, ×500, hematoxylin stain, BP120.)

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were taken under ordinary light microscopy; (B, D, and F) under phase contrast. (Hematoxylin stain.)

(A and B) Normal corneal endothelium. The intercellular cement substance is clearly seen in (B) the phase-contrast picture. The cornea was stained with alzarin red before fixation in formalin (BP158).

(C and D) The displaced nuclei appear rod, dumb-bell, or sickle-shaped (BP121).

(E and F) Pigmented granules, clumps of pigment, and a pair of abnormally large nuclei overlie the globular body, which is seen in the phase contrast picture (F) as a laminated, less dense round body (BP159).

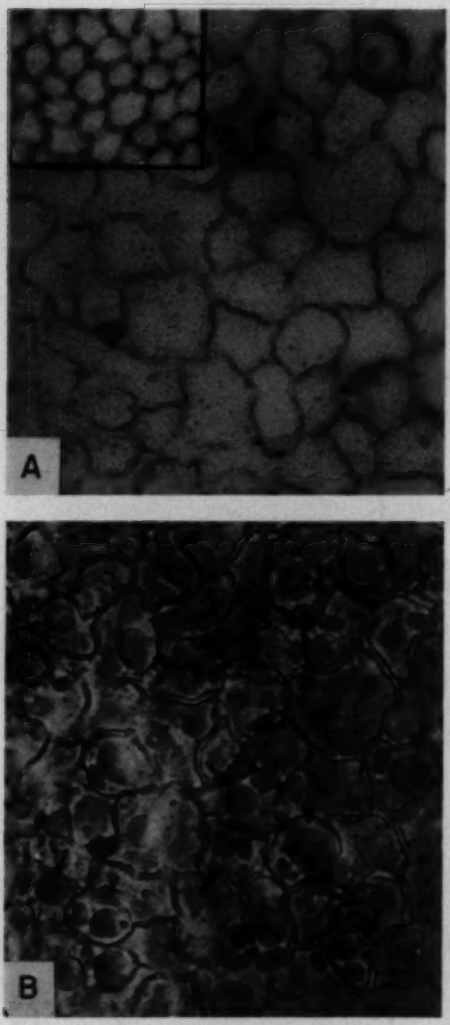

Fig. 4 (Chi, Teng, and Katzin). Fresh, unfixed cornea, stained with alzarin red to show the intercellular cement substance (×160, BP160).

(A) Irregular pattern of endothelial cells in endothelial-epithelial dystrophy. The inset in the upper left-hand corner shows the regular pattern of normal endothelial cells.

(B) The globular bodies of various sizes indicated by the arrows are seen under phase microscopy to be intracellular, cytoplasmic bodies.

lular and intercellular edema (fig. 15). This is followed by bulla formation both subepithelially and intraepithelially (figs. 16, 17, and 18). Intraepithelial bullae occur less often than subepithelial bullae. The bullae are filled by a coagulated or fibrillar mass (figs. 16, 17, and 19).

Occasionally a characteristic, laminated avascular fibrous tissue is seen between Bowman's membrane and the epithelium (fig. 20). The corneal epithelium is very irregular, both as to number of layers of cells and as to the shape and appearance of the individual cells. In places the thickness may be seven or more cells; in other places it is reduced to two or three cells (figs. 15, 16, 17, and 18).

The basement membrane and Bowman's membrane are present over most of the area, but in places they may be absent. Generally speaking, Bowman's membrane seems to be fairly well preserved (figs. 16, 19, and 20).

Comment

In view of the description just given, the essential histopathologic findings in endothelial-epithelial corneal dystrophy appear primarily in the endothelium. Changes in Descemet's membrane, the stroma, and epithelium seem to be secondary.

A review of the literature shows that the mechanism producing these changes is not thoroughly understood. It is generally accepted that Descemet's membrane is a product of the endothelium. There is considerable evidence that Descemet's membrane can regenerate if the endothelium is present but it does not reform in the absence of endothelium. This has been demonstrated experimentally and is seen in pathologic conditions. It is also a well-known fact that Descemet's membrane grows thicker with advancing age (fig. 21).

Using special stains Descemet's membrane appears to be composed of two layers, an anterior elastic layer which does not seem to change its thickness throughout life, and a posterior mucoprotein layer which increases its thickness with age (fig. 21). In babies' eyes the posterior layer of Descemet's membrane is not present (fig. 21-A and B).

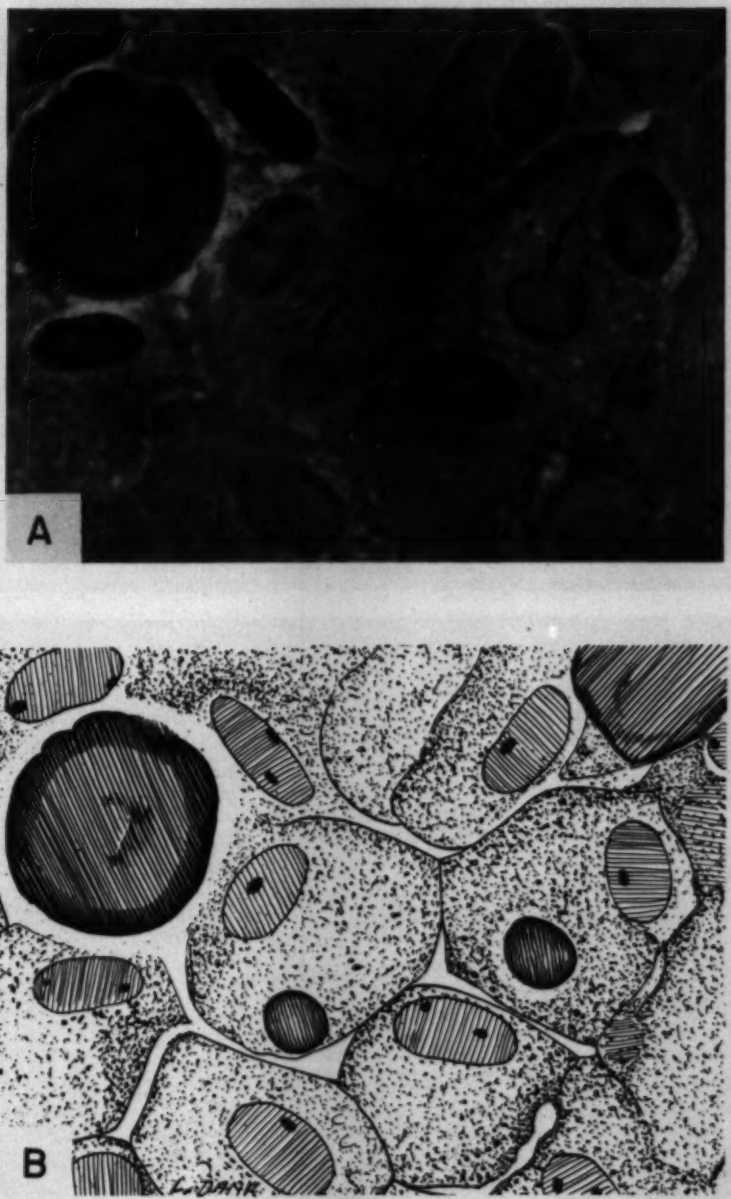

Fig. 5 (Chi, Teng, and Katzin). (A) The endothelial cells are larger and their nuclei contain nucleoli. The two small globular bodies indicated by the arrows appear to be intracellular, cytoplasmic bodies. (Flat preparation, phase contrast, ×500, hematoxylin stain, BP 122.) (B) Diagrammatic sketch of A.

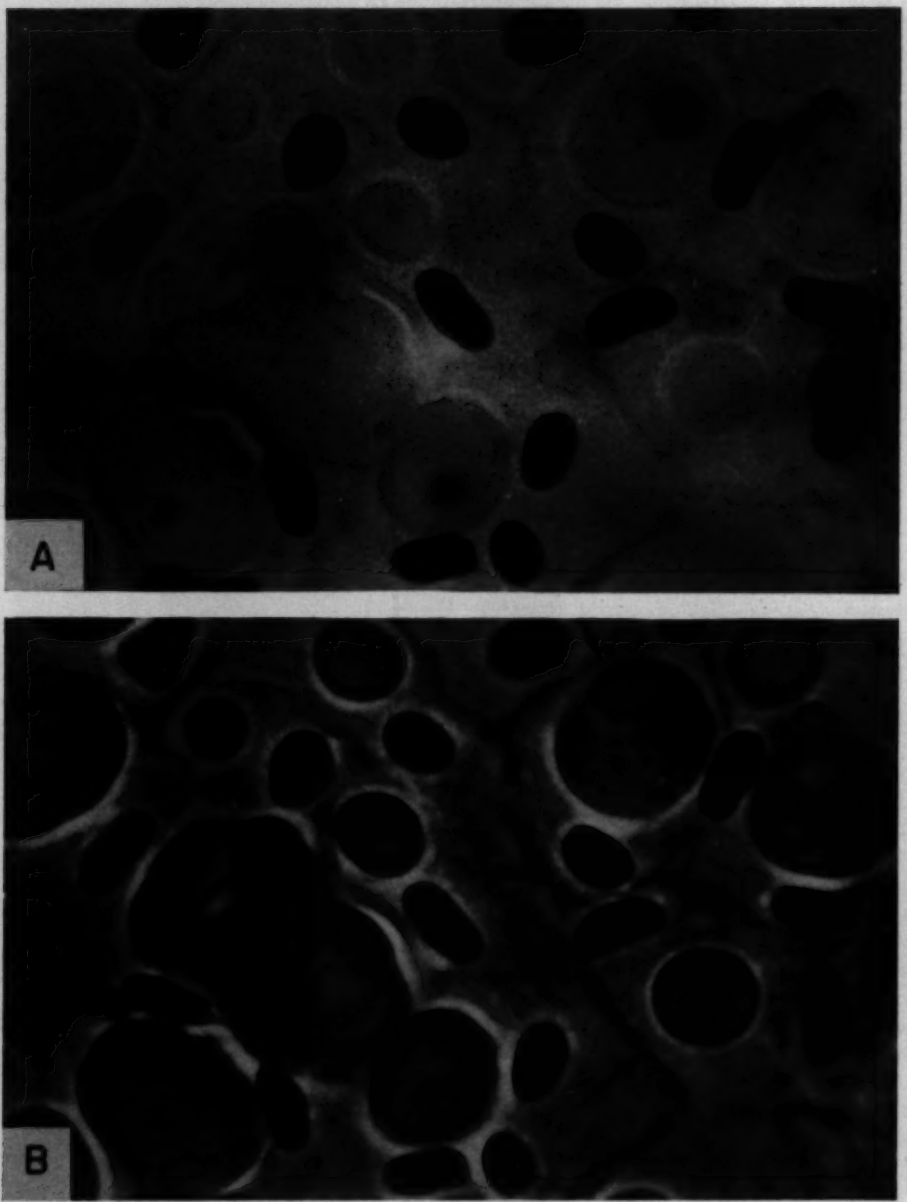

Fig. 6 (Chi, Teng, and Katzin). Three globular bodies have broken through their cellular boundaries and fused. Arrows in picture (B) (phase-contrast) indicate globular bodies which seem to be cytoplasmic inclusions, with eccentric nuclei. (Flat preparation, ×500, hematoxylin stain, BP160.)

Fig. 7 (Chi, Teng, and Katzin). In cross section the globular bodies appear as nodular excrescences on Descemet's membrane (D). The endothelial cytoplasm (E) is thinned out over the top of the excrescences. (Cross section, ×375, periodic acid-Schiff stain BP124-12.)

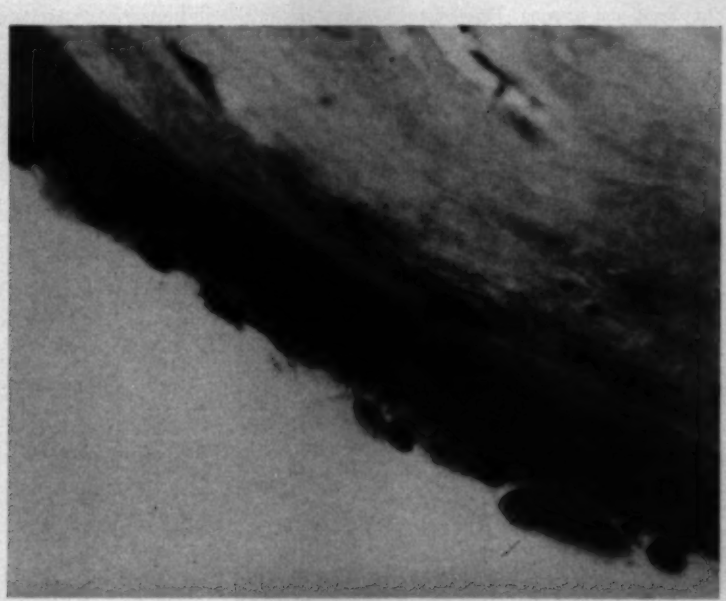

Fig. 8 (Chi, Teng, and Katzin). Nodular excrescences on Descemet's membrane (D) with the endothelial nuclei pushed toward the valleys between the excrescences. (Cross section, ×375, periodic acid-Schiff stain, BP125-17.)

Fig. 9 (Chi, Teng, and Katzin). The nodular excrescences on Descemet's membrane are ovoid or anvil-shaped. Here a narrow dark anterior layer of Descemet's membrane is apparent. (Cross section, ×250, Verhoeff's stain, BP 154-3.)

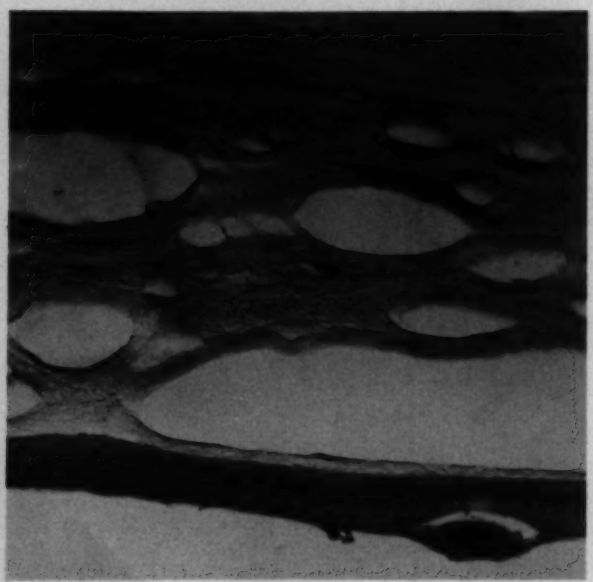

Fig. 10 (Chi, Teng, and Katzin). One of the nodular excrescences appears to be separated from Descemet's membrane. It shows its biconvexity. (Cross section, ×250, periodic acid-Schiff stain, BP122-16.)

Fig. 11 (Chi, Teng, and Katzin). The globular bodies appear as intracellular cytoplasmic inclusions (indicated by arrows). The cytoplasm contains fine pigment granules. The nuclei show distinct nucleoli. (Thin flat section, ×500, periodic acid-Schiff stain, BP125-9.)

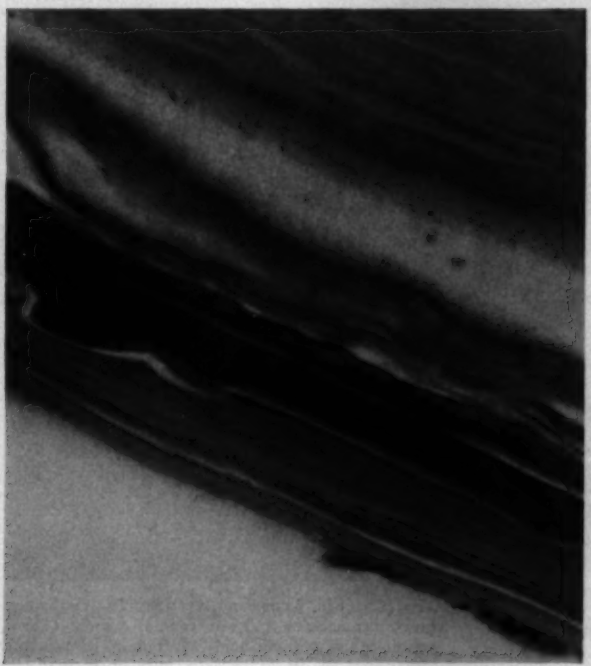

Fig. 12 (Chi, Teng, and Katzin). An additional laminated membrane overlies the nodular excrescences on the original Descemet's membrane. A few elongated nuclei of regenerated endothelium line this new membrane. (Cross section, ×375, hematoxylin and eosin stain, BP159-13.)

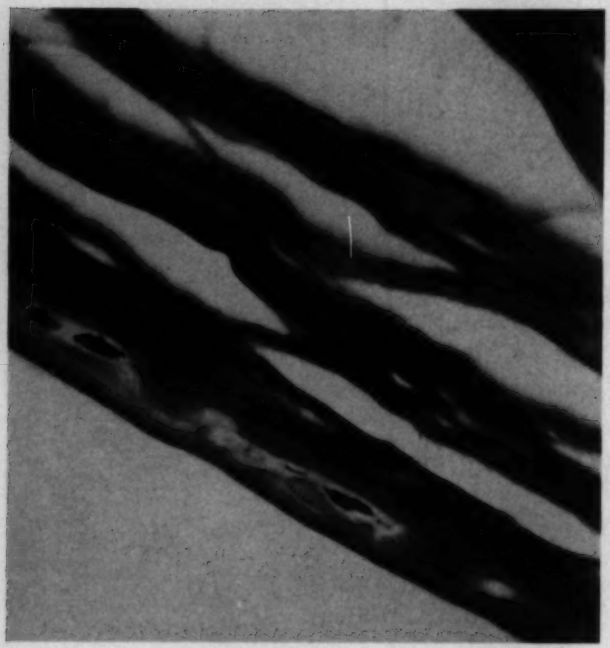

Fig. 13 (Chi, Teng, and Katzin). Clumps of pigment granules lie between the nodular excrescences on the original Descemet's membrane and the new overlying membrane. (Cross section, ×375, toluidine blue, BP138-5.)

Fig. 14 (Chi, Teng, and Katzin). Fibrous tissue is seen between the epithelium and the fairly well-preserved Bowman's membrane (B). The anterior stroma has separated into fine, light fibrillae with loss of metachromasia. (Cross section, ×150, toluidine blue stain, BP138-5.)

Fig. 15 (Chi, Teng, and Katzin). The epithelium shows intracellular and intercellular edema. (Cross section, ×150, periodic acid-Schiff stain, BP126-16.)

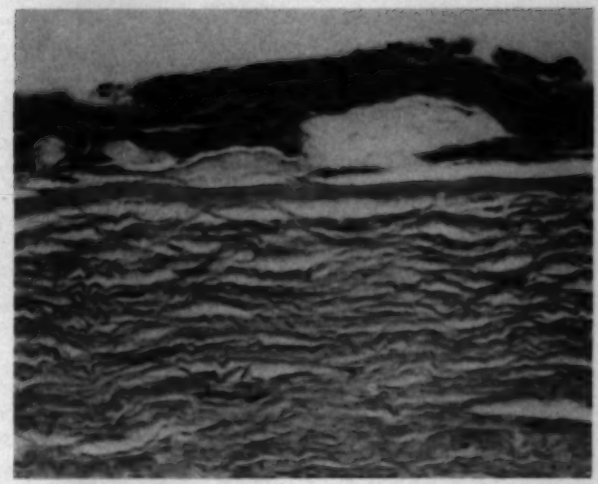

Fig. 16 (Chi, Teng, and Katzin). Bullae of various sizes have formed between the epithelium and Bowman's membrane. They are filled with coagulum or fibrillar tissue. The cell layers of the epithelium are irregular. (Cross section, ×150, hematoxylin and eosin stain, BP142--6).

In flat preparations, cross and flat serial sections reveal that the initial lesion appears as a cytoplasmic body in the endothelial cells (figs. 4-B, 5, and 11). These cytoplasmic bodies may be the result of hyperactivity of the endothelial cells, or a product of intracellular metabolism, or they may be phagocytosed substances. The nature of these cytoplasmic bodies is still not well known. Further histochemical studies and detailed

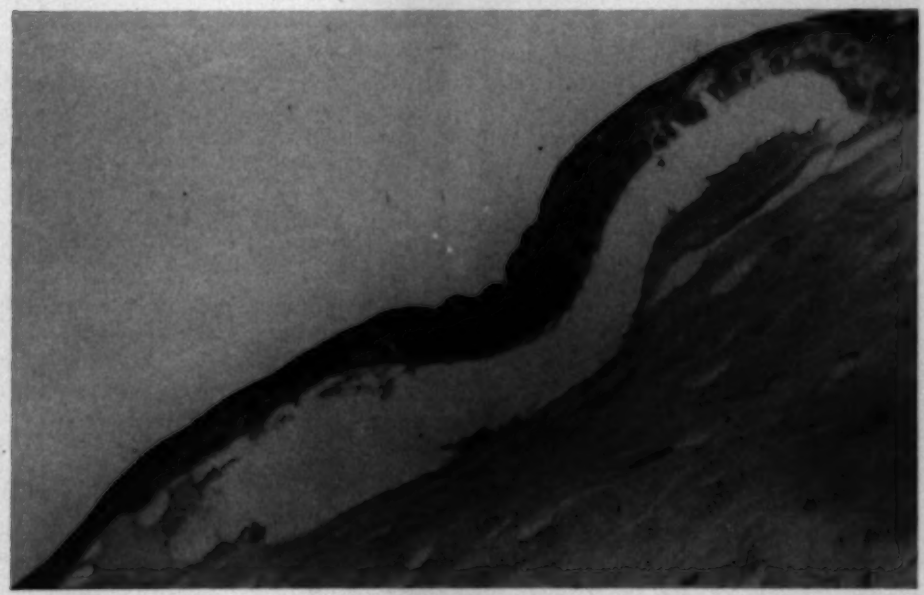

Fig. 17 (Chi, Teng, and Katzin). The cell layers of the epithelium are irregular and a large bulla lies between the epithelium and Bowman's membrane. (Cross section, ×150, periodic acid-Schiff stain, BP126-5.)

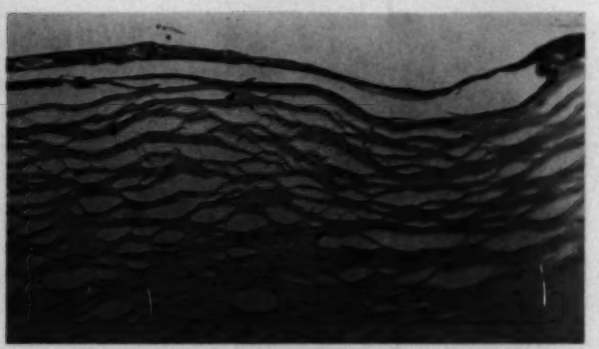

Fig. 18 (Chi, Teng, and Katzin). A large intraepithelial bulla and edema of the anterior layers of the stroma. (Cross section, ×56, periodic acid-Schiff stain, BP126-5.)

observations of their character using the electron microscope are contemplated.

In the early stages, the endothelial cells with cytoplasmic bodies appear larger and their nuclei contain distinct nucleoli (figs. 5 and 11). As these cytoplasmic bodies increase in size, they coalesce anteriorly with Descemet's membrane and form nodular excrescences (figs. 7, 8, 9, 10, and 12). Posteriorly the cytoplasm of the endothelium thins out over the top of the excrescences (fig. 7), and laterally the nuclei are displaced to the sides of the excrescences (fig. 8), re-

Fig. 19 (Chi, Teng, and Katzin). A fibrillar tissue (F) has formed between the epithelium and Bowman's membrane (B) which appears fairly well-preserved. (Cross section, ×375, hematoxylin and eosin stain, BP159-13.)

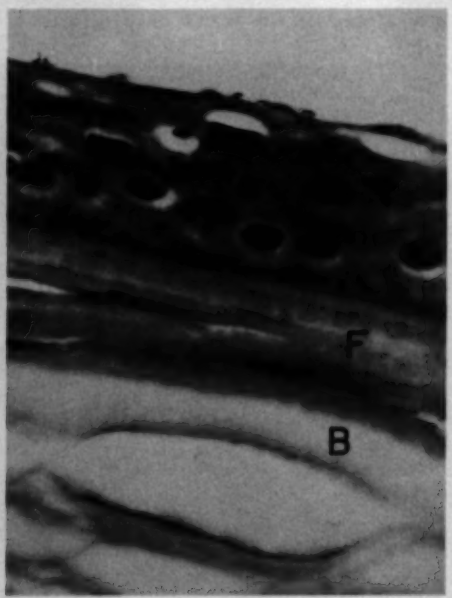

Fig. 20 (Chi, Teng, and Katzin). A laminated, avascular fibrous tissue (F) is seen between the epithelium and Bowman's membrane (B). The anterior stroma is edematous. (Cross section, ×375, periodic acid-Schiff stain, BP159-13.)

sulting in a distortion of the cells and their nuclei. Eventually the endothelial cells burst and are disintegrated.

Verhoeff* and Stocker[10] each reported histologic studies of an eye with endothelial-epithelial corneal dystrophy in which there was duplication of Descemet's membrane without excrescences. Calhoun's[18] case showed nodular excrescences and duplication of Descemet's membrane. In our series of 20 cases we found four cases such as the one described by Calhoun and some areas of three other cases were similar.

In flat preparations seen under the phase contrast microscope, the globular bodies appear to be covered by a layer of substance and cells with abnormally large nuclei (figs. 1-F and 2-F). In cross section one can definitely see a new membrane and large, elongated nuclei overlying the original Descemet's membrane with excrescences in between (fig. 12).

Although in hematoxylin and eosin stain there may sometimes appear to be duplication of Descemet's membrane without intervening excrescences, nevertheless excrescences may be found when the specimen is examined under the phase contrast microscope. What appears to be a diffuse thickening of Descemet's membrane in this stain is seen by phase contrast study to be closely packed globular bodies which have fused together.

Degeneration and regeneration of the endothelial cells sometimes seem to occur simultaneously. A difference in the rate of the repair process may account for the two different histopathologic pictures of this disease. In cases where there are excrescences on Descemet's membrane without duplication of the membrane, the degeneration of the endothelium is very extensive and there is no chance for the endothelium to regenerate.

In the rare cases where there is duplication of Descemet's membrane, we believe

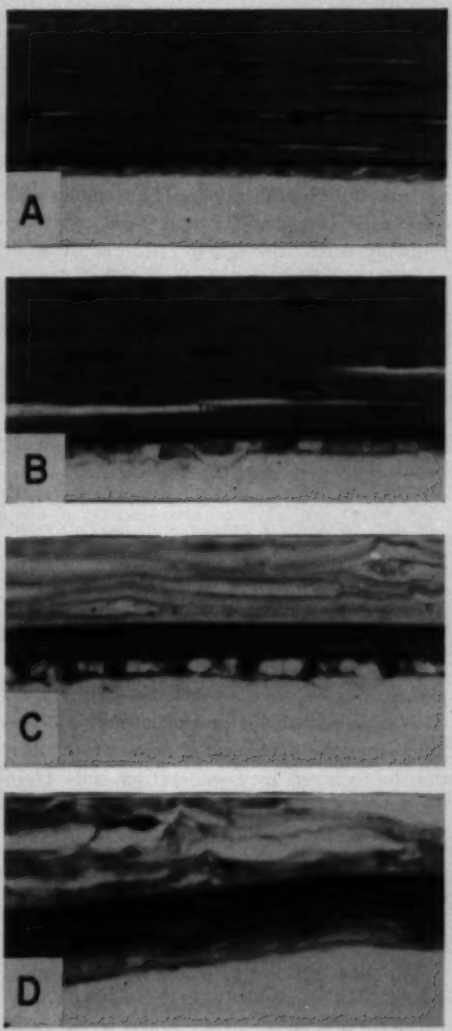

Fig. 21 (Chi, Teng, and Katzin). Descemet's membrane in normal eyes of various ages compared with Descemet's membrane exhibiting endothelial-epithelial dystrophy. Verhoeff's stain and the same magnification (×250) in each picture.

(A) Normal Descemet's membrane of stillborn, six-month fetus (EB2504).

(B) Normal Descemet's membrane in one-day-old, full-term baby (EB4827).

(C) Normal Descemet's membrane in 59-year-old woman (EB3910-76).

(D) Descemet's membrane in endothelial-epithelial dystrophy of a 57-year-old patient (BP137-8).

*Lloyd reported histologic studies by Verhoeff.

that the repair process may be under way. The regenerated cells are often seen in what appear to be isogenous groups as shown in Figures 1-E and F and 2-E and F, and it may be assumed that mitosis or amitosis has occurred. We especially looked for cells in mitosis but found none. The regenerated cells with large nuclei and faint staining probably occur through the process of amitosis from adjacent normal endothelial cells and migrate to the degenerated areas to cover them, eventually forming a new membrane.

In Figure 12 a definite lamination of the new membrane is distinguishable. This condition is certainly evidence that the membrane was produced by regenerated endothelium. The arrangement in layers is possibly due to several episodes of secretion or regeneration.

The clinical course of this disease probably also depends on the extent of regeneration of the corneal endothelium. Clinically the disease process is so slow that many cases never advance beyond the early stages. This makes it all the more reasonable to assume that the degenerated areas are continuously repaired by regenerating cells from the adjacent normal cells.

The epithelial and stromal changes are secondary to the degeneration of the endothelium and occur only when this degeneration has become extensive enough to allow the access of aqueous into the cornea.

We share Stocker's[16,17] and Paton's[18] opinion that keratoplasty may be successful in cases of this type of dystrophy, if the cases are properly selected; that is, if there is a fair amount of healthy peripheral endothelium present. The graft should be large enough to include all the definitely diseased parts of the cornea, so that these parts are replaced by healthy tissue.

No light has as yet been thrown on the etiology of this condition. Most authors agree with Verhoeff*[19] who stated that these phenomena are the result of secretion by hyperplastic or abiotrophic endothelium undergoing senile or presenile degeneration. Clinically it may occur as congenital, familial endothelial dystrophy as reported by Theodore.[20] Unfortunately there was no histologic examination of his cases, although their slitlamp appearance very much resembled the presenile or senile type.

It is unanimously agreed that women are much more frequently affected than men, which may be an indication of an unknown additional factor. It is possible that some abnormal stimulus or substance is present in the aqueous, which renders the endothelium capable of producing the cytoplasmic bodies. The aqueous of the eyes and the systemic condition of the patients should be studied and correlated with other findings.

In flat preparation of the corneal endothelium examined under phase contrast microscope, the cytoplasmic inclusions or globular bodies almost dominate the field (fig. 1-D). The appearance closely resembles the slitlamp picture of the endothelium seen clinically, except that under the ordinary magnification of the biomicroscope the nuclei of the cells cannot be seen clearly.

Finally we want to emphasize the value of the phase-contrast microscope in the study of the corneal endothelium and the nodular excrescences on Descemet's membrane. These phenomena can be beautifully demonstrated by this method. Whereas, changes in the endothelium may be difficult to see and evaluate in cross serial sections, they may present strikingly abnormal changes in flat preparation under phase-contrast microscopy. Phase-contrast microscopy is also invaluable in correlating the histopathologic picture with the clinical, slitlamp picture.

Summary and Conlusions

Twenty of the 28 cases examined proved to be primary endothelial-epithelial corneal dystrophy and the report on these 20 cases is added to the previous 11 cases reported

* Verhoeff, F. H.: In discussion of paper by Levitt and Lloyd.

in the literature. The mechanism producing the disease is presented and its etiology is discussed. Flat preparations of the corneal endothelium examined with the phase-contrast microscope make possible a much better correlation of the clinical and pathologic findings.

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Ophthalmic Miniature

The endeavor to belittle v. Graefe's merit in reference to glaucoma simply because he did not lay the same stress on the separate symptoms as we do at the present day, reminds me of the lectures of a teacher who never became tired in demonstrating to his pupils how round-about was the course taken by Columbus when trying to discover America.

A. Mooren,
"Remarks on the development of glaucoma,"
Archives of Ophthalmology, **13**:15, 1884.

DELAYED RECURRENCES OF MALIGNANT MELANOMAS OF THE BULBAR CONJUNCTIVA*

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AND

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Malignant melanoma of the bulbar conjunctiva is one of the rarest of ocular neoplasms. This statement is as true today as it was in 1903, when Verhoeff and Loring[1] documented it with a survey of the world literature. Another observation of these authors is also true today: "Unfortunately the end-results have been but seldom noted, so that accurate data as to recurrences and permanent cures are wanting."

At the turn of the century several writers cited by Verhoeff and Loring were of the belief that these were relatively benign tumors, which, although prone to recur locally, rarely penetrated the globe and seldom metastasized. Verhoeff and Loring did not share this opinion, and consequently were opposed to simple excisional therapy. Since then, epibulbar melanomas have generally been regarded as highly malignant.

That such a fearful outlook is not entirely justified is suggested by the follow-up study of 49 patients with epibulbar melanomas in the Registry of Ophthalmic Pathology, summarized by Ash in 1950.[2] Twenty-seven patients (55 percent) were known to be living without evidence of recurrence or metastasis; 16 of them had been followed five years or longer. In a current study of the registry material (as yet not completed), the junior author has been impressed by the long survival in certain cases and by the great variability in patterns of recurrence and metastasis. The senior author has had under his care one of the most remarkable of these cases, one in which local recurrence was not observed until almost 18 years after enucleation. After a brief review of the pertinent literature, this case will be reported in detail, and synopses of other cases germane to our discussion will be given.

REVIEW OF LITERATURE

INCIDENCE

In 1903, Verhoeff and Loring[1] culled 73 cases from the literature. At their own institution, the Massachusetts Charitable Eye and Ear Infirmary, conjunctival melanomas had been seen in only two of 44,719 patients examined during a two-year period. Crigler[3] reviewed a series of 100,000 conjunctival lesions which included 100 tumors, but only four of these were malignant melanomas. Ball and Lamb[4] reported on the pooled data of several workers: there were 14 epibulbar malignant melanomas in a population of 671,242 patients with ocular complaints. Rifat[5] reported three cases of conjunctival melanoma in 18,495 patients. Benedict[6] summarized the experience at the Mayo Clinic between 1917 and 1936. Only 11 tumors of the bulbar conjunctiva were observed among 138 malignant melanomas of the eye.

Reese[7,8] was of the opinion that the occurrence of malignant melanoma in a conjunctival nevus was much more rare than cancerous change in diffuse conjunctival melanosis, for he could find only two of the former in the voluminous literature up to the year

* From the Registry of Ophthalmic Pathology, Armed Forces Institute of Pathology, Washington, D.C. Presented at the 93rd annual meeting of the American Ophthalmological Society, Hot Springs, Virginia, May, 1957. This paper will be published in the *Transactions* of the American Ophthalmological Society and is printed here with the permission of the society and of Columbia University Press, New York, New York.

† By invitation. From the Central Laboratory for Pathologic Anatomy and Research, Veterans Administration, Armed Forces Institute of Pathology.

1941. The more limited experience of Swan and his associates[9] was confirmatory.

In reviewing the registry material, one of us (L. E. Z.) has been impressed by the frequency with which a spot had been present for many years before the malignant change occurred. Verhoeff and Loring[1] cited six cases in which sudden growth and extension were observed in small spots that had been present for periods of 10 to 30 years, and two cases in which the spots had been noted for "many years."[10-17] Tweedy[18] mentioned a case he had seen in which a lesion had remained the size of a pinhead for more than 20 years before it became malignant. Crigler[3] summarized two cases in which nevi became malignant after dormant periods of 12 and 20 years. Swan and associates[9] described two cases in which nevi observed in infancy became malignant 20 and 47 years later, but neither of these was at the limbus.

It has also been noted that the histologic features of epibulbar melanomas are often indicative of a pre-existing benign nevus.[19]

From 1940 through 1955, there were 270,000 admissions to the city of Memphis hospitals, about 4,000 for ocular disease but only one was for an epibulbar malignant melanoma. During the period 1944-1956, not one malignant melanoma of the conjunctiva was encountered in a series of 20,000 patients examined in the eye clinic of the Memphis Eye and Ear Hospital.

Hogan[20] stated that only one, a malignant melanoma near the limbus, had been seen among 36,000 patients examined at the University of California Eye Clinic during a three-year period.

The senior author has seen only two such melanomas among private and clinic patients during his 33 years in the practice of ophthalmology. The junior author has had an opportunity to review more than 100 cases in the Registry of Ophthalmic Pathology of the Armed Forces Institute of Pathology, submitted by ophthalmologists and pathologists from all over the world during a period of more than 35 years. A better sense of the proportion in which these tumors occur is conveyed by the fact that for every epibulbar malignant melanoma in the registry there are roughly 40 intraocular melanomas.

Recurrences

Recurrence is known to be a common sequel of incomplete removal of conjunctival melanoma. Even the early writers cited by Verhoeff and Loring[1] were impressed by this tendency, even though they did not regard the epibulbar melanoma as highly malignant. Among the 73 cases collected by Verhoeff and Loring, local excision was the treatment in 53. Only two were without recurrence during follow-up of several years: Joerss'[21] patient, followed for four years, and Weinbaum's[22] for 10 years after the second local excision. Among the 36 cases with known recurrence, there were three in which it became manifest after more than five years, including the case of Panas[23] in which the tumor recurred 10 years after removal.

In discussing recurrence following local excision or enucleation, a distinction should be made between two basically different types: (1) Recurrence that represents spread from the original tumor, either before the operation, or by seeding of tumor cells during local excision or enucleation; (2) recurrence that may be a completely new focus of neoplasia.

The existence of multicentric foci of carcinogenesis has become widely recognized in a variety of malignant tumors. This distinction is seldom made in the literature on recurrence of conjunctival melanoma. However, Reese's cancerous melanosis is a good example of a tumor in which multiple foci of malignant change are to be expected, while malignant melanoma arising from a pre-existing benign nevus is a focalized phenomenon, not associated with other sites of melanoma formation.

In 1928, Veil[26] presented the case of a 73-year-old woman with a "nevocarcinoma" of the limbus. After biopsy and radium treatment, the tumor was excised widely. For six

months the patient considered herself cured but the tumor recurred and metastasied. This author also mentioned two other patients with epibulbar melanomas whom he had treated successfully by excision (four-year follow-up). All three tumors were similar microscopically. In discussing treatment, Veil took the fatalistic view that wide excision should suffice for even extenteration will not be curative if metastasis has occurred. He believed the prognosis of a progressive but still freely movable melanoma to be favorable if excision is performed early and completely. Tumors that were adherent to the cornea and sclera he considered more ominous and, in such cases, recommended electrolysis, galvanocauterization, or diathermy coagulation of the tumor bed after excision.

The rationale of Veil's approach is essentially the same as for exenteration. It is assumed that with clinical signs of infiltration, there probably are deep extensions of tumor into the conjunctival lymphatics, the sclera, or even the uvea. In this regard Dennis,[27] in a recent paper, has quoted Verhoeff as having advised the compromising procedure of removal of "a portion of the ciliary body if necessary."

Treatment

At the time of Verhoeff and Loring's paper, the tendency was to do simple excisions and to proceed with more radical operations only after recurrences made them absolutely necessary. Verhoeff and Loring, however, were opposed to such practice for they believed it jeopardized the patient's life. These authors recommended enucleation, as did Duke-Elder[24] who added that as much conjunctiva should be included as possible, and "if necessary evisceration of the orbit, with subsequent radiation." He did not elaborate on how to decide when evisceration becomes necessary. Morax[25] also believed the danger of dissemination to be sufficiently great to call for wide excision and sacrifice of the eye.

In our present state of relative ignorance and in view of our inability to predict accurately which tumors will recur and metastasize after excision and enucleation, it is probably wise to follow Reese's[8] advice to proceed with exenteration of the orbit as soon as an unequivocal histopathologic diagnosis of malignancy is established. Such a practice is based on the realization that it is impossible to ascertain whether or not local extensions or lymphatic spread have gone beyond the immediate vicinity of the visible tumor.

On the other hand, experiences such as we are about to recount, in which patients survived many years with or without local recurrence after simple excision, demand that we re-evaluate surgical management of these limbal melanomas.

Case reports

AFIP accession no. 47994

On October 6, 1935, a white woman, 34 years of age, was referred by her family physician, Dr. J. A. McQuiston, because of a growth of the right eyeball, present for three or four months. There had been no previous eye trouble of any kind and her general health had always been excellent.

Examination showed a tumor of the conjunctiva of the inferomedial quadrant which extended six mm. along the limbus and posteriorly for nine mm., from the 4- almost to the 6-o'clock position. The growth was about one mm. in thickness. Its surface was smooth except for a small eroded area. Its color was salmon pink and it was supplied by several large blood vessels. The tumor could be moved with the conjunctiva except at the limbus where it was fixed. It did not invade the clear cornea. Otherwise the eye was normal as was also the left eye. There were no enlarged lymph nodes.

On October 7, 1935, the tumor was excised with a surrounding border of apparently normal conjunctiva. The underlying sclera and the corneoscleral junction were cauterized with the actual cautery and the entire defect was covered with sliding flaps of conjunctiva. Microscopic examination of the tissue by a local pathologist revealed a "nonpigmented malignant melanoma, probably not completely removed, at the limbus." On October 10, 1935, enucleation and implantation of a glass ball were performed. A good cosmetic result was obtained and the patient wore a Snellen reform eye with complete comfort for almost 18 years. She was examined frequently during the first five years, after that, only occasionally.

On August 17, 1953, the patient came to the office complaining that the socket had been sore

for almost two weeks. A rounded, firm, slightly elevated, reddish mass, about four mm. in diameter, occupied the medial third of the socket. The conjunctiva was intact, there being no erosion of the surface. No secretion was present and the regional lymph nodes were not palpable. The following day the nodule was excised along with a wide margin of normal conjunctiva.

The tumor was examined histologically by Dr. W. W. Tribby, pathologist of the Methodist Hospital and the Memphis Eye and Ear Hospital. He reported that it was a highly malignant, nonpigmented melanoma. Three days later, on August 20, 1953, the orbit was exenterated. No tumor tissue nor cells were found in the excised mass.

The cavity healed slowly but completely. No attempt was made to fit a prosthesis. The patient usually wears a white patch; sometimes an opaque lens with a side shield. Her physical condition has continued to be excellent as has also her morale. She leads a busy life, being active and useful in her home, church, and community. Up to the time of this report there have been no metastases.

The original tumor, the enucleated globe, and the recurrent tumor were sent to the Armed Forces Institute of Pathology. All have been restudied microscopically by one of us (L. E. Z.). The histopathologic observations are as follows:

Biopsy of 1935. The conjunctival nodule is covered by greatly attenuated epithelium, one large area of which is completely permeated by tumor cells and ulcerated (figs. 1 and 2). The intraepithelial portion of the invading tumor includes a considerable number of cells that contain a moderate amount of golden brown pigment. Most of the tumor cells are nonpigmented and epithelioid in appearance, with large pleomorphic nuclei, many of which have prominent nucleoli (fig. 3).

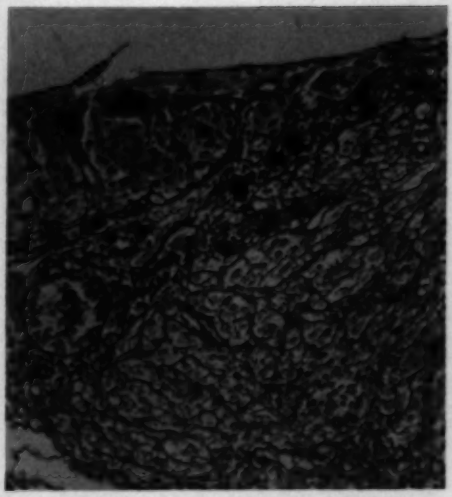

Fig. 2 (Lewis and Zimmerman). Same field as Figure 1 stained by the Wilder technique for reticulum. Masses of melanoma cells replace the conjunctival epithelium and invade the subepithelial tissues. (×230.)

Along one margin of the nodule, interlacing bundles of spindle-shaped cells predominate. These, too, are mostly nonpigmented. In several foci, the spindle and epithelioid elements are intermingled. Along the deeper margins of the specimen there are foci of lymphocytic infiltration. The original nevoid pattern of the tumor is completely effaced

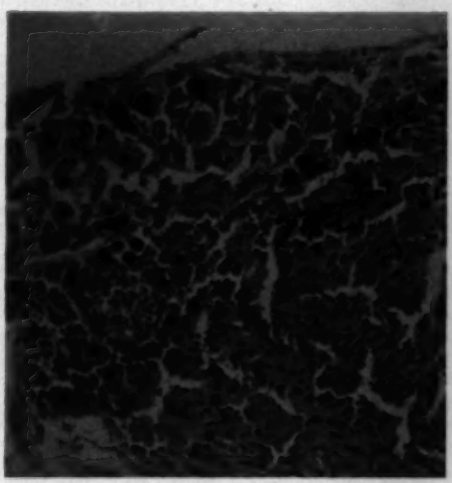

Fig. 1 (Lewis and Zimmerman). Malignant melanoma of bulbar conjunctiva excised in 1935. (Hematoxylin-eosin, ×230.)

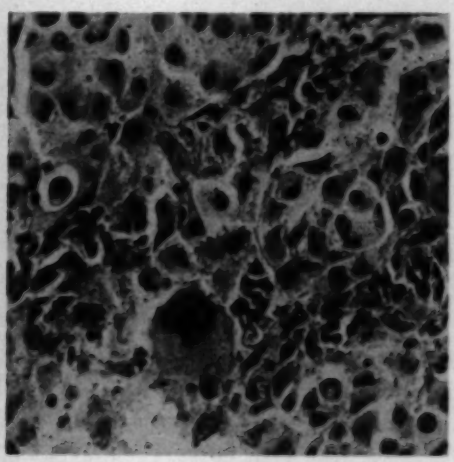

Fig. 3 (Lewis and Zimmerman). Pleomorphic epithelioid cells of the malignant melanoma excised in 1935. (Hematoxylin-eosin, ×440.)

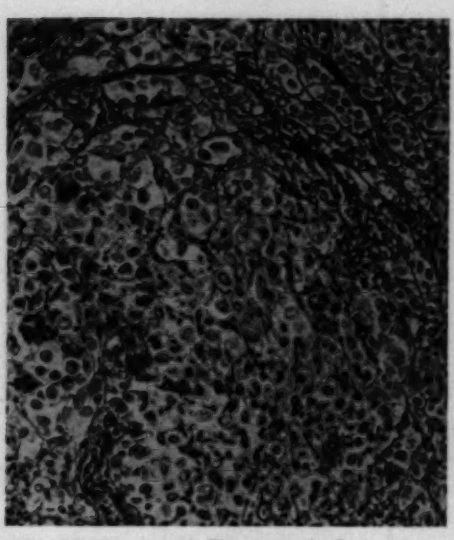

Fig. 4 (Lewis and Zimmerman). Loss of organoid pattern, invasiveness, and paucity of reticulum fibers about the large epithelioid cells are demonstrated in this section stained by the Wilder technique. (×245.)

by the diffusely infiltrating tumor cells. In spite of the obvious anaplasia, mitotic figures are few.

The Wilder stain for reticulum fibers accentuates the invasive properties of the tumor and reveals but few foci in which the tumor cells are surrounded by a well-developed argyrophilic reticulum. For the most part, only nests and lobules of neoplastic cells are surrounded by reticulum fibers (figs. 2 and 4).

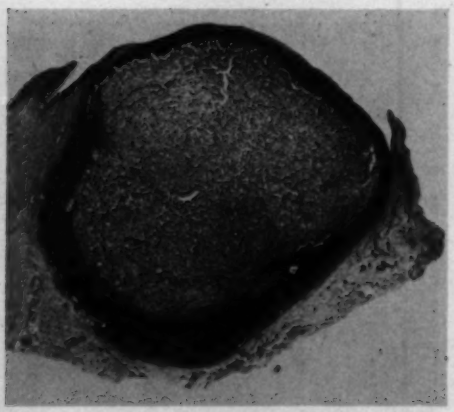

Fig. 5 (Lewis and Zimmerman). Recurrent malignant melanoma excised from conjunctiva of socket in 1953. A very thin layer of atrophic but otherwise uninvolved epithelium covers the tumor nodule which is completely surrounded by an infiltrate of lymphocytes. (Hematoxylin-eosin, ×15.)

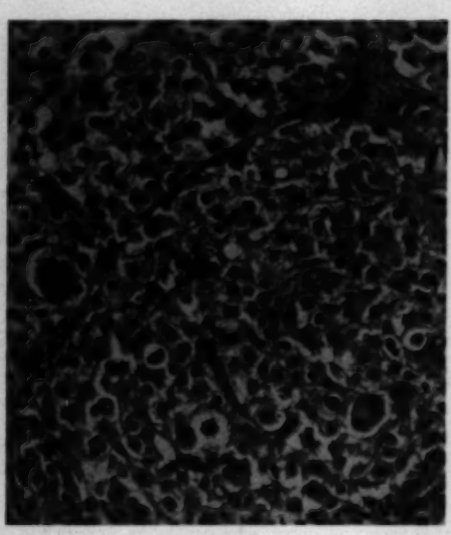

Fig. 6 (Lewis and Zimmerman). The recurrent tumor is comprised almost entirely of nonpigmented pleomorphic melanoma cells of epithelioid type. (Hematoxylin-eosin, ×160.)

Diagnosis. Malignant melanoma, bulbar conjunctiva, mixed cell type.

Microscopic description of enucleated globe. The bulbar conjunctiva at the site of excision of the tumor nodule is partially re-epithelialized but a larger part is covered by a deposit of fibrinous exudate infiltrated by a moderate number of polymorphonuclear leukocytes. Proliferation of fibroblasts and capillaries and the presence of thrombosed vessels are observed along the episcleral surface. Although a number of bizarre proliferating fibroblasts are noted throughout the granulation tissue, definite evidence of tumor is not found. The intraocular structures appear to be normal.

Diagnosis. Essentially normal eye; granulation tissue at site of recent excision of conjunctival tumor.

Tumor removed in 1953. The tissue excised from the conjunctiva lining the socket contains a spheroidal nodule which lies just beneath the conjunctival epithelium which itself is not involved (fig. 5). A zone of dense lymphocytic infiltration forms a mantle circumscribing the tumor. Occasional reactive centers are seen within this lymphocytic infiltrate. The tumor is composed almost entirely of large, pleomorphic, and frequently bizarre neoplastic cells (fig. 6). Aggregations of spindle-shaped cells are seen in several foci. Huge multinucleated forms and mitotic figures are numerous and dispersed throughout the nodule. Occasional dilated spaces lined by epithelium and filled with

mucus are present in both the tumor and the lymphoid tissue that surrounds it. Although some of the tumor cells are faintly stippled with golden brown pigment, the majority appear completely amelanotic.

Diagnosis. Recurrent malignant melanoma of socket.

Comment. The recurrent tumor is similar to that excised from the bulbar conjunctiva in that it is composed predominantly of amelanotic melanoma cells, both spindle and epithelioid. The recurrent tumor differs from the primary in its greater proportion of epithelioid cells, which, in general, are larger and more pleomorphic than those observed in the original lesion. Mitotic figures are also more numerous in the recurrent tumor.

The question might be raised as to whether the tumor excised from the socket in 1953 represents a new primary conjunctival melanoma or a metastasis which had remained dormant since 1935. The observation of an intact layer of uninvolved conjunctiva covering the nodule and the layer of lymphocytes interposed between the conjunctiva and the nodule are against its being a new primary. It is generally conceded that primary melanomas of the skin and mucous membrane arise within the epithelium, or at least they have a prominent junctional component. Metastatic tumors of the skin and mucous membrane, on the other hand, are devoid of junctional activity. Involvement of the epithelium as observed in the primary lesion excised in 1935 was completely lacking in the sections prepared from the specimen excised in 1953.

A review of the literature and a survey of the registry material failed to produce a case comparable to the one we have just described. However, several in the registry bear on the general problem of the natural behavior of epibulbar melanomas; six of these have been selected for brief presentation.

AFIP ACCESSION NO. 52160

A 38-year-old white woman had conjunctival tumor which was first observed five years before the present admission. Excisions had been performed four years and two years previously. The eye was enucleated on January 18, 1937. Examination of the specimen revealed an epibulbar mass, 3.0 by 2.0 by 2.0 cm., extending from the cornea to the equator. Microscopically, the neoplasm was a malignant melanoma of mixed cell type, showing great variation in pigmentation. In October, 1939, a mass attached to the tarsus of the upper eyelid was excised. Microscopically, this proved to be a recurrence of the original tumor. In January, 1956, 23 years after the first operation and 16 years after the last, the patient was examined by Dr. Hedwig S. Kuhn who found no evidence of recurrence or metastasis.

AFIP ACCESSION NO. 35659

A 44-year-old, dark-skinned Negro woman presented herself in 1931 with a large vascular mass protruding through the lids from the bulbar conjunctiva of the right eye. A spot had been present since infancy and for one year it had been growing rapidly. The mass finally "ruptured" and bled profusely. A clinical diagnosis of corneal staphyloma was made and the eye was enucleated on August 13, 1931. Microscopic examination revealed only granulation tissue. The contributing ophthalmologist reaffirmed that the patient was a "full-blooded Negro" and furnished preoperative and postoperative photographs for documentation.

In January, 1939, the patient was seen by another physician because of a tumor of the thigh. Aspiration biopsy revealed metastatic malignant melanoma. Radiation therapy was administered, the patient having refused amputation. However, more than a year later she returned, and the leg was amputated through the hip joint on May 10, 1940, almost nine years after enucleation of the eye. Information supplied by Dr. W. R. Mathews of Shreveport, Louisiana, indicates that the patient was followed and was known to be "free from recurrence of her disease" as of March, 1956.

AFIP ACCESSION NO. 60859

A 51year-old woman submitted to enucleation of her right eye because of a recurrent tumor of the limbus. Eleven years earlier another ophthalmologist had excised a growth which, though attached to the globe, protruded through the lids sufficiently to touch the cheek. According to the patient it was about an inch in length. Following excision, cauterization was performed eight or nine times in the course of 18 months. It was not ascertained whether the original tumor had been examined microscopically. At the time of enucleation in October, 1938, the cornea and limbus were thickened and scarred inferiorly. Microscopic examination of the eye revealed recurrent, lightly pigmented malignant melanoma at the limbus, infiltrating the corneal stroma. The patient was known to be alive and without recurrence or metastasis in October, 1946.

AFIP ACCESSION NO. 554064

The left eye of an 80-year-old white woman was enucleated on October 13, 1952, because of the presence of a lobulated, pigmented mass, 13.5 by 8.0 by 5.0 mm., at the limbus in the lower nasal quadrant. A lesion had been excised in November, 1944, and biopsy was performed on a recurrent lesion in 1951. Microscopic examination of all three specimens revealed malignant melanoma of the bulbar conjunctiva. When last examined by her ophthalmologist, Dr. E. G. Nadeau, in November, 1956, the patient was still in excellent health. She had had two small local recurrences, the second of which was "pea-sized" when excised in September, 1956. Exenteration has not been recommended because of the patient's age.

AFIP ACCESSION NO. 67398

A 71-year-old white woman was seen in February, 1940, because of an elevated, brownish-white, infiltrative lesion at the limbus in the lower nasal

quadrant. It extended into the cornea for three or four mm. Some "congenital pigment" was observed in the adjacent bulbar conjunctiva. The vessels supplying the tumor were engorged and increased in number. The lesion was excised on February 20, 1940. Microscopic examination revealed a malignant melanoma, but conservative management was recommended since it was believed that "these melanomas at the limbus are not nearly so malignant as those of the uveal tract."

The patient returned in September, 1942, with another lesion at the lower temporal limbus of the same eye. It was excised on November 23, 1942. Pathologic diagnosis again was melanoma of the conjunctiva.

In March, 1948, the patient returned with a more elevated, cauliflowerlike tumor at the limbus between the 5- and 7:30-o'clock positions. It was excised on March 30th, but this time the tumor was more difficult to strip away and it seemed to infiltrate Bowman's membrane and the stroma. Microscopic examination revealed a more anaplastic form of malignant melanoma. The neoplasm recurred promptly, extending from the 4- to 8-o'clock positions. Exenteration was performed on April 22, 1949. The specimen received in the laboratory revealed an epibulbar tumor, 30 by 24 by 16 mm. Microscopically it was a very anaplastic and amelanotic malignant melanoma, composed of plump spindle and pleomorphic epithelioid cells.

The patient was last seen by the contributing ophthalmologist in January, 1950, but is known to have died subsequently. As yet, we have been unable to ascertain the cause of death.

Lest the pathologist be criticized too severely for having recommended conservative treatment of the original limbal tumor in the case just reviewed, we wish to cite the following example of a good result obtained by simple excision.

AFIP ACCESSION NO. 710505

A 70-year-old white man was first seen by Dr. Warren A. Wilson in December, 1939, because of a black mass, 1.0 by 1.0 cm., which covered approximately two thirds of the cornea and projected between the lids. The tumor apparently had developed in the course of 18 months before simple excision was performed on December 26, 1939. Although a pathologic diagnosis of malignant melanoma was made, the patient refused either enucleation or exenteration. The patient returned to Dr. Wilson in February, 1955, for refraction. Except for arteriosclerotic Parkinson's disease, he was considered in good health for his age of 85 years. The original sections of the melanoma have been reviewed; the tumor is still considered histologically malignant.

DISCUSSION

The cases presented emphasize the need for good clinicopathologic studies with long periods of follow-up. As there is not a single such study recorded, we do not possess sufficient factual data upon which to base conclusions regarding surgical management. The cases recorded here were obviously selected to illustrate the fact that long survival is possible in spite of repeated recurrences and conservative surgical measures. Admittedly it is dangerous to draw conclusions from selected cases, but one should, nevertheless, learn from his own experience.

The senior author has had two patients with epibulbar melanoma. One was treated with excision followed by enucleation. The second was treated by simple excision and the patient remains well with an intact normal eye seven years later. He believes, therefore, that if the tumor is so situated that excision can be made with a fairly wide margin (two to three mm.) and if upon microscopic examination is appears to have been completely excised, further surgery is not indicated until recurrence is evident. This is particularly true in the case of individuals with only one good eye and in very elderly patients. Following excision the patient should be examined carefully at frequent intervals. If the tumor extends widely or infiltrates the corneal or scleral stroma, enucleation, and in some instances with more extensive disease, exenteration will be required.

CONCLUSIONS

Malignant melanoma of the bulbar conjunctiva is a rare tumor, the natural behavior of which is as yet not understood. A thorough clinicopathologic study with long-term follow-up of a large series of such cases is needed before valid conclusions can be drawn regarding proper management. The possibility that this tumor may be adequately treated by simple excision still deserves consideration.

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Erratum

The Journal deeply regrets that the color plate (frontispiece, accompanying paper by Rones, B.: A mechanistic element in trabecular function. Am. J. Ophth., **45**: facing p. 189 [Feb.] 1958) was printed upside down. The legends are, therefore, reversed: Figure 6 should be Figure 1; 5 → 2; 4 → 3; 3 → 4; 2 → 5; 1 → 6. The Journal apologizes to Dr. Rones and to its subscribers.

POSTOPERATIVE CARE OF RETINAL DETACHMENT*

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In an address at Hamilton, Ontario, in February, 1956, Sir Francis Fraser,[1] director of the British Postgraduate Medical Federation and professor emeritus of medicine, University of London, said: "In clinical medicine it is rare for the evidence to be sufficient to justify a conclusion on scientific grounds, but it would be quite unjustifiable on that account to suspend judgement and its consequent action. The situations that the doctor has to deal with commonly in clinical medicine cannot then be dealt with wholly by the methods and criteria of science and he has to bring to his aid a practical art. . . . I doubt if the foundations of this aspect of medicine have altered since the days of Hippocrates."

My approach to this subject is somewhat philosophical and eclectic. Unsupported by finished experimental and laboratory studies, my thesis is this: cure follows good surgery in cases of retinal detachment regardless of posture and attempts at fixation.

It is natural, when high authority points the way, to follow dutifully. This may or may not be commendable. Immobilization has been the rule for years, and there is a natural resistance of human attitudes to change. However, one is less than wise, if, as he goes along the well-worn way, he fails to explore those paths that promise a quicker climb or lead through fairer fields. The eye is never motionless, even in sleep, and actual fixation would require elimination of all stimuli, which is practically impossible.[2] If prolonged attempted immobility after surgery for retinal detachment is to be continued, let us have some proof that it is necessary. To paraphrase an apt comment from one of my correspondents, let us not in this case submit to an immobilization of our thinking.

My opinions were first publicly expressed[3] at the November, 1951, meeting of the Southern Medical Association. Subsequently, a report was published in the January, 1952, *Archives of Ophthalmology*,[4] and again in January, 1953, in The American Journal of Ophthalmology.[5]

Some eight years ago I operated on an aphakic eye for retinal detachment in an elderly man with heart disease who could not lie down. He was not put to bed nor restricted in any way. The retina healed in place. At that time, I was unaware that Duke-Elder,[6] and Arruga,[7] and perhaps others, are lenient in such cases. If this man could get well, I thought others should, and so I began cautiously to allow freedom of motion and soon was convinced that reasonable bodily activity is probably irrelevant to cure.

I do not take the position that we can be careless in handling these patients. On the contrary, each deserves our most serious and meticulous attention. I have no unalterable rules. However, in general it may be said that no effort is made to control posture; there are little or no restrictions on toilet privileges, diet, or catharsis; peephole glasses are not always used; both eyes are kept closed; pressure is not used; the operated eye is observed on the third day; a chair is allowed from the third day on; moderate sedation is used for the first three days; the unoperated eye is left open on the fifth day; the patient goes home in five to seven days; atropine is used for three weeks; and the patient is back at work in four to six weeks.

Robert A. Brown and I have cared for 55 eyes, a number having been operated on more than once. We have refused only one case the chance of help by at least one opera-

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* Presented at the 93rd annual meeting of the American Ophthalmological Society, Hot Springs, Virginia, June, 1957. This paper will be published in the *Transactions* of the American Ophthalmological Society and is printed here with the permission of the society and the Columbia University Press, New York.

tion. Surgery has been done on at least eight eyes which appeared hopeless to begin with, and of these one became reattached. Three cases have been classified as failures in which detachment recurred from three to six months after an apparent success. We have not been impressed with full-thickness scleral resection and prefer lamellar resection with some form of scleral folding, electrocoagulation, and fluid evacuation as a primary procedure in all but the simplest and most favorable cases, of which we have seen few. We have had no occasion to regret our procedures of after care and have procured apparently permanent reattachment in slightly more than 50 percent of all cases.

Thinking that it would be of interest to know what is generally being done in this connection, I wrote a personal letter some months ago to 70 individuals having board certificates; not all, however, practicing in large metropolitan areas. I asked for information as to how each managed retinal detachment cases postoperatively, as I was interested in the subject. Comments were invited. There were 57 replies from 28 states and four foreign countries. Some avoided definite answers. Eight recommended bedrest for less than a week; 36 for one to two weeks; 10 more than two weeks. Two emphasized that eye rest is more important than body rest. Two stated that they do not use peephole glasses. Those using peepholes use them for two weeks or longer, usually much longer.

Unpublished work by Clark[8] on dogs has shown that adhesive strength between retina and choroid after scleral cauterization is present at 36 hours. This is probably true in the human eye but yet unproved. Although my patients are up on the third day, I do not presume to say that healing is complete in 72 hours. On the contrary, it is certain that the full process requires weeks or months.[9] The facts in the case are not known. However, Dunnington's and Clark's large experience[10] and my limited one bear out my contention.

What immediate effect cauterization has on the tissues involved is also unknown. It may be that the choroid is so stimulated or irritated as to promote absorption of retinal fluid in cases where the retina is not flat at the end of surgery. I will yield that in such cases a few additional hours, or even days, of rest may be of value, but only in the event that the area of the hole has not been brought in apposition to the pigment epithelium and sealed there with the cautery, a process not requiring coagulation to the extent that it can be observed ophthalmoscopically.

Once sealed in place, it is difficult for the retina to leave its pigment epithelium for, if the retina separates, the space created must be filled with something. Detachment cannot therefore be suddenly produced except by the most violent kind of direct trauma or by hemorrhage. This I have had demonstrated to me when, in passing a hypodermic needle into the vitreous for the purpose of air injection, I have seen the retina advance in front of the point for several mm. in an extremely sharp tent formation, finally rupture, and with remarkable elasticity return with a snap to its natural position.

Whether the retina is in proper position or not, it is, in the absence of vitreous adhesions, subject to the same pressure from within as from without, since the eye as a whole is a solid body, and even after surgery with fluid release, relatively normal pressure is quickly re-established by the intraocular secretory mechanism. It is, therefore, quite possible that the disturbed vitreous, quite fluid in the usual case of detachment,[11] slips as harmlessly over the surface as does water over the sides of a glass which is rotated in the hand. Even in the area of the hole, the effect of a passing fluid on a very delicate edge, subject to equal pressure on both sides, would seem so small as to be ineffectual in altering, in either direction, relative positions, except possibly by repeated rhythmical and unavoidable movements, such as occur with the eyes closed or even in sleep.

This is more particularly true if coagulation has been used to strengthen the natural though tenuous bond existing between the retina and its pigment epithelium. It seems quite possible that the minute and frequent vibrations transmitted through the vocal chords may be more harmful than the beautifully balanced and tension-free motions of voluntary rotation for visual purposes.

One might reasonably ask at this point whether peephole glasses are at all desirable in these patients. Certainly, if they are used, fixation through the aperture is necessary, and in the process of fixation there is an inherent and irregular[12] muscular effort.[13] This results in movement of two kinds, large jerky agitation of which the subject is aware, and fine subconscious quivering. When the eye is steadiest, it maintains one position not longer than one fifth of a second.[14] Accompanying movements of the head must also be taken into account. It is entirely possible that the use of peepholes may add to rather than subtract from the difficulties. In the absence of proof to the contrary, it is just as well to relieve the patient of this cumbersome burden.

It is not possible to consider here all the complications that can result and have resulted from prolonged efforts at immobilization. It is conceivable that such attempts can stimulate fundamental psychologic elements of fear and anger, and so alter hormonal activity and emotional[15] stability as to be detrimental to reparative processes.

The patient with retinal detachment is usually a high-strung, emotional, tense person, and immobility does not necessarily reduce bodily tension.[16] Indeed, in the absence of emotional tranquility, rest may even increase tension.[17]

How much importance do the emotions have in the cure of this condition? Have you not all observed and wondered that the patient who steadfastly believes in his recovery often does recover against apparently insuperable difficulties? This whole subject is replete with interesting and at present unanswerable questions.

There are patients who simply refuse surgery in the face of prolonged immobilization. A near relative is a case in point. He actually died with a blind eye because he would not consent to such treatment with its attendant difficulties.

Finally, and least important, though in some cases a deciding factor, is the matter of finance. Some patients will go on with one good eye and refuse surgery rather than assume the heartbreaking expense of long convalescence. If a short hospital stay can be established as adequate, it will mean a great deal to many individuals, and to those organizations devoted to blind prevention and rehabilitation.

In conclusion, relative freedom in the postoperative management of retinal detachment is thought to be a sound and proper procedure.

101 Church Street.

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AN EXPERIMENTAL INVESTIGATION OF THE BASIC PHENOMENA OF RETINOPEXY*

Part I. Electrical impedance measurements

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Introduction

This report constitutes one in a series of reports on the results of a three-year study on the basic phenomena of electrical energy in the treatment of retinal detachment. The second report will cover the measurement of heat developed in the ocular tissues during such treatment and the thermal effects on the tissues. Some of the practical implications have been outlined in a recent paper by Irvine and Knoll.[1]

In order to appreciate fully the phenomena involved in electrothermally produced tissue damage, a brief review of alternating current principles will be presented. A very concise statement of these principles has been published by Hemmingway and Stenstrom.[2] The section which follows represents a summary of this excellent report.

Alternating current concepts

The force which drives electrons through an electrical circuit is called the electromotive force (E). The unit of electromotive force is the volt. The rate of flow of electrons per unit time represents the current (I) and the unit is called the ampere. The various components of a circuit tend to impede (under special circumstances enhance) the current flow. The unit of impedance (Z) is the ohm.

The basic components of an alternating circuit are resistors, condensers, and coils. The impedance to the current flow in these components is referred to as resistance, capacitive reactance, and inductive reactance respectively. In direct current circuits only the first of these plays a role.

Electromotive force, current, and impedance are related by Ohm's law which states that the current flow is directly proportional to the electromotive force and inversely proportional to the impedence. Mathematically the relationship is written thus:

$$I = E/Z$$

The current and electromotive force will vary in magnitude with time. Heating effects are expressed in terms of the effective values which are defined as the values which are equivalent to the values of direct current and electromotive force values. The relationship between maximum and effective values is as

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following in systems involving sinusoidal currents

$$I_{eff} = 0.707\ I_{max}$$
$$E_{eff} = 0.707\ E_{max}$$

The power in watts (P) developed by an electrical current flowing in a conductor is given by the product of the effective current and the effective electromotive force, thus:

$$P = I_{eff}\ E_{eff}\ \text{Cos}\ \Theta^*$$

Written in terms of current and impedance this relationship may be written as follows:

$$P = I^2_{eff} Z\ \text{Cos}$$

and in terms of electromotive force and impedance:

$$P = E^2_{eff}/Z\ \text{Cos}\ \Theta$$

The work done in time t shows up as heat and it is this heat which results in tissue damage when tissues are a part of the electrical circuit.

The heat equivalent in terms of the above is given by P t (0.241) = calories delivered in time t, expressed in seconds.

The resistive component of impedance is independent of the frequency of the alternating current. The capacitive reactance is inversely related to frequency, whereas the inductive reactance is directly related to frequency. Since tissues act as though they contained resistance and capacitive reactance, tissue impedances vary inversely with the frequency.[3]

Electrocoagulation and Electrical Impedance

Measurements of tissue impedances have been carried out for most of the bodily tissues[4, 5] and more recently Meyer-Schwickerath[6] has measured the impedance of the individual ocular tissues dissected from freshly enucleated human eyes. Since electrode impedances were carefully avoided the values obtained represent the impedance of the tissues per se. Meyer-Schwickerath measured the ocular tissue impedances in the frequency range 0.1 to 100 million cycles. He found that the humors had a fairly constant impedance over the frequency range measured. The impedance of the other ocular tissues varied most at the lowest frequency and all approached the value for the fluid contents at the highest frequency. These findings are in accordance with our knowledge of tissue impedances in general, namely that the capacitive reactance arises at the cell membrane and hence the tissues with the highest cell density (retina and lens) will demonstrate the largest impedance variation with frequency.

Since it was the purpose of the present study to duplicate actual operative procedures as closely as possible the values given by Meyer-Schwickerath's study served only as a starting point for our work.

During electrocoagulation for retinal detachment (and also in other ocular electrocoagulation procedures) several sources of electrical impedance are present. This impedance may be made up of various combinations of the following:

1. Resistance in the leads;
2. Capacitive reactance between the leads, and the leads and ground;
3. Inductive reactance in the leads;
4. Resistance and capacitive reactance of the ocular tissues which lie between the treated eye and the indifferent electrode, and
5. Capacitive reactance arising at the point of contact between each of the electrodes and the tissues.

Each of these sources has been evaluated and the results will be presented later.

Method and Results

Impedance measurements were made at six frequencies between and including 100 cycles per second and 10 million cycles per second. A Hewlett-Packard Test Oscillator, Model 650A, was used to generate the energy at these frequencies. The output was connected across a known resistance in series

* Θ represents the phase angle between the current and the voltage. It may vary between zero and 90 degrees. Cos Θ is referred to as the power factor.

with the experimental animal. Contact with the animal was made in the following manner. A pointed electrode needle was placed in contact with the animal's sclera and the contralateral ear was grounded through a saline soaked gauze pad and a flat copper clamp. The output of the oscillator was adjusted until a voltage drop of one volt existed across the known resistor and the animal. The voltage drop across the animal was then measured. The impedance could then be calculated from the known value of the resistor and the voltage drops. The voltage measurements were made using a General Radio vacuum tube voltmeter Type 1803-A.

The anesthesia used in all the experiments was nembutal injected intrapleurally. The dosage used was 30 mg./kg. of body weight.

The results for untreated dog sclera, plotted as open circles, may be seen in Figure 1. It will be noted that the impedance is high at the low frequencies and falls off in a more or less linear manner as the frequency increases. This decrease in impedance with increased frequency is in agreement with our knowledge of tissue impedance.

Measurements of impedance were also taken after surface treatment and puncture. The equipment used to treat was a Walker Combination Galvanic and Diathermic Unit. The dial settings used are indicated on the figures, as for example, W50. The duration of the treatment is also indicated in seconds following the dial setting. The number in parentheses indicates the number of measurements averaged to arrive at the values plotted. Note that the impedance does not change appreciably after surface treatment. The impedance does drop after puncture. This drop in impedance is felt to be due to the increased contact between needle and tissue after puncture.

Figure 2 shows the same type of data for rabbit sclera.

In order to determine the impedance of tissues lying beneath the sclera an insulated flat electrode having a circular cross section of 0.885-square mm. surface area was used as the active electrode.

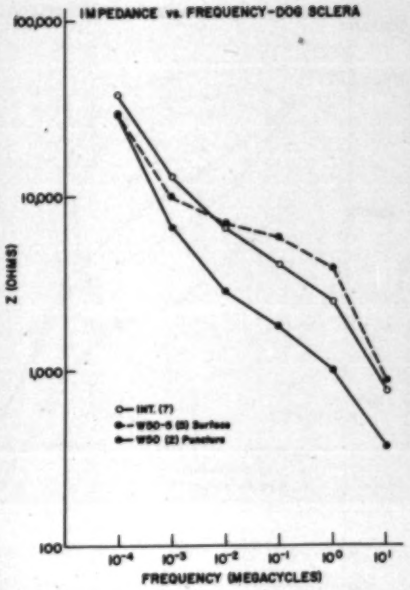

Fig. 1 (Knoll). Impedance as a function of frequency. Pointed active electrode placed directly on the sclera. Ground electrode on contralateral ear using saline-soaked gauze sponge and copper clamp. The three curves represent measurements taken on fresh sclera, following surface treatment and following puncture. The figures in parentheses represent the number of determinations averaged for the point plotted. W50 refers to the dial setting of the Walker Unit and the number five included for the surface treatment indicates that the treatment was of five seconds' duration.

This electrode was placed (a) on the sclera, (b) directly on the choroid, after dissecting away sclera, and (c) in the vitreous about five millimeters below the inner surface of the choroid. The same type of ground electrode was used as previously.

These data are plotted as open circles in Figure 3. There seems to be no significant difference between the impedance of sclera and choroid, but the impedance of the vitreous does seem to be lower at all frequencies.

To test the effect of changing the ground electrode, the ground lead was attached to a lid speculum placed beneath the lids of the eye which is contacted by the active electrode. These data are also plotted in Figure 3. Here again there seems to be little difference

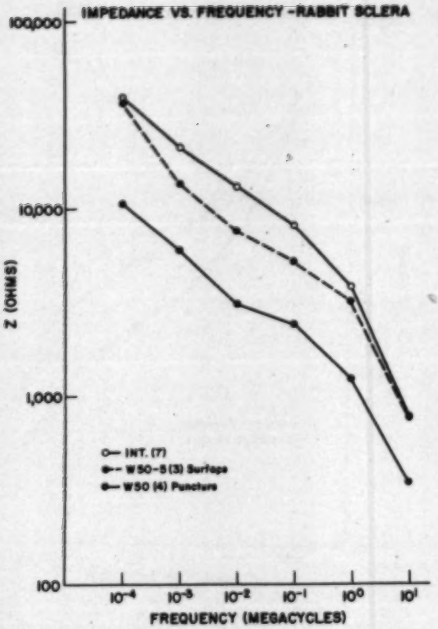

Fig. 2 (Knoll). Same as the first figure using the rabbit as the experimental animal.

between sclera and choroid and a large difference when it comes to vitreous. Note also the variation with respect to change in ground lead. One must conclude that a considerable impedance does arise at the point of contact of the ground electrode and that this effect is greatest at the lower frequencies.

To test the contribution of the tissues intervening between the electrodes hypodermic needles were placed in the muscle tissue of the contralateral forelimb and in the muscle tissue of the contralateral hindlimb. When these were used as ground electrodes the impedance values did not vary significantly. Hence it may be concluded that the body tissues contribute a very small portion of the impedance at least as compared to the impedance at the electrodes.

Varying the length of the leads from six inches to six feet, twisting and coiling the leads did not lead to significant changes in impedance.

Measurement of impedance during actual treatment

In the measurements thus far described an effort was made to avoid tissue damage while the impedance measurements were carried out. It was for this reason that the voltage across the tissues was kept at approximately one-half volt. Do these values of impedance apply during actual treatment? The tissue fluids will be heated to the boiling point and the intraocular pressure rises considerably and certain physical and chemical changes will take place in the tissues surrounding the tip of the active electrode. In order to investigate the impedance during treatment the cornea and sclera of cats were treated with currents having the following frequencies, 6,000 cycles, 1.5 megacycles, and 10 megacycles. An Army Signal Corps transmitter was modified to supply power at these fre-

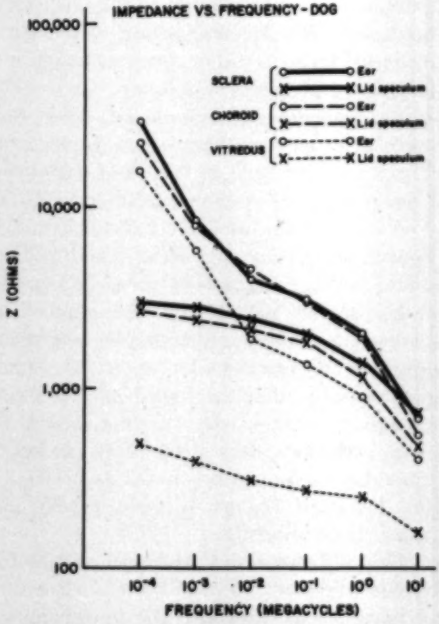

Fig. 3 (Knoll). The impedance of sclera, choroid, and vitreous using two types of ground contact on the animal. Each point represents the average of two determinations taken on different animals.

quencies at 50 volts. The currents flowing during three-second treatments were observed on a thermocouple ammeter.

During the duration of the treatment it was found that there was an initial surge of current lasting about one half second. The current then settled to a stable value for the remaining two and one half seconds. Using these values of current and the fixed voltage of 50 volts, the impedances were calculated.

The surge impedance values thus obtained agree very closely with the values shown in Figure 3 for sclera and choroid using the lid speculum (the lid speculum was used in these experiments as the ground electrode). The impedance values calculated once the current has dropped to its stable value gave results that were 3.5 times higher at 6,000 cycles, three times higher at 1.5 megacycles, and 1.2 times higher at 10 megacycles. It is suggested that the rise in impedance after the initial half second results from the boiling away of tissue fluid. This point will be more fully discussed later.

When the active electrode was allowed to puncture and hence come in contact with the vitreous or aqueous, very high values of current were recorded. In many cases the ammeter needle went off scale. The impedance values thus obtained approximated the values given in Figure 3 for the vitreous using the lid speculum.

Discussion

Electrically, one may think of cell membranes as "leaky" condensors, that is, the membrane acts as an almost perfect insulator. The fact that it is not perfect allows some current to pass, although presenting a very high resistance. The extra- and intracellular fluids may be represented as resistances. The schematic electrical model may then be thought of as shown in Figure 4. This type of circuit will show a decrease in impedance with an increase in frequency, although not as shown by any of the relationships in Figures 1, 2, or 3. The reason for this discrepancy lies in the fact that the

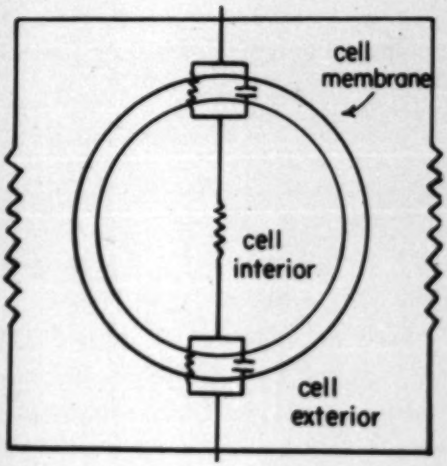

Fig. 4 (Knoll). Electrical model of a single cell surrounded by tissue fluid.

values of capacitance and resistance are probably not fixed from frequency to frequency. That this is the case in lysed erythrocytes has been shown by Schwan and Carsensen.[7] It would simplify matters if these values did remain constant since then one could easily calculate the power absorbed by the tissues at each of the frequencies.

The results of the experiments in which the impedance was measured during treatment would suggest that here, too, the values of capacitance and resistance may change. It is felt that the change during treatment comes about largely as a result of the vaporization of the extracellular fluid which initially provided the path of least impedance to the current flow, particularly at the lowest frequency.

This relationship is emphasized by the marked decrease in impedance when the cellular layers are pierced and the electrode comes into contact with the vitreous and aqueous.

Electrically the ocular tissues become more and more homogeneous as the frequency of the current increases. The danger of large variations in heating would be accomplished if equipment were used having frequencies above 10 megacycles.

Summary

1. Impedance measurements were made on the eyes of dogs and rabbits at six frequencies between and including 100 cycles per second and 10 million cycles per second. In all experiments the impedance was highest at the lowest frequency and decreased with increasing frequency.

2. Impedance measurements following surface treatment with a Walker Unit indicated little or no change in impedance. Measurements made following puncture indicated a lowering of impedance. The drop of impedance may be due to increased surface contact or to contact with vitreous which has an impedance significantly lower than choroid and sclera.

3. Impedance with electrode contact on sclera and choroid are equal within experimental error for all frequencies measured. Impedances with electrode contact in the vitreous are lower by a factor of one tenth.

4. Variations in impedance were found as a result of using different types of ground electrodes, particularly at the lower frequencies.

5. Variation in the distance between active and ground electrodes had no significant effect on the impedance. Neither did the length and configuration of the leads.

6. During surface treatment with a sine wave oscillator at frequencies of 6,000 cycles and 1.5 and 10 megacycles at 50 volts, the initial impedance agrees with the experimentally determined values, but rises to a higher value after about one-half second of treatment. The increase in impedance is inversely proportional to the frequency.

7. During puncture treatment the impedance drops from the stable value by a factor of 50 to 100. This value of impedance agrees approximately with that found for the vitreous.

8. These effects can be explained by an analogous electrical circuit whose component values vary with frequency and treatment conditions.

Department of Biophysics (24).

Acknowledgment

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RETINAL SUTURES*

An experimental evaluation for treatment of detachment of the retina

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The idea of applying retinal sutures for the treatment of detachment of retina is not entirely new. If one pursues the annals of scientific venture, one finds that almost nine decades ago this idea was conceived but, probably on the grounds of insufficient evidence and conservatism, it was lost in oblivion.

A closer scrutiny of the literature motivated the undertaking of this experimental study. It was felt by us that the ideas embodied in the past literature needed further investigation and study.

Historical survey

Meyer in 1871,[1] as cited by C. A. Wood,[2] and quoted by Krewson,[3] was the first to conceive the idea of passing a suture through the retina and drawing it to an opening in the scleral wall.

Ribard,[4] in 1876, tried a gold suture in eight cases and reported that there was "tolerance parfaite" in most of the cases. DeWecker and Grizou,[5] in 1877, attempted to establish a more permanent form of drainage by passing a curved needle with a fine gold thread through the sclera and choroid and out again and tying the ends over the conjunctiva, but the eyes were lost due to infection and irritation.

Rubbrecht,[6] in 1933, tried to produce retinochoroidal adhesions by placing 0-0 silk sutures in the eyes of rabbits and leaving them for two days, with satisfactory results. Then sutures were tried in human eyes lost because of old detachment of the retina. No unfavorable reaction was produced. Finally he tried two patients with recent detachment of the retina. They were operated upon by placing two sutures at the border of the detachment and leaving them for two days. In each case reattachment was obtained, with useful vision.

Present study

The materials employed in this study were plain gut 5-0, chromic gut 6-0, silk 6-0, and nylon 6-0.

Technique

Rabbits of average weight were anesthetized by intravenous pentobarbital sodium. A couple of drops of pontocaine (0.5 percent) were instilled in the eye. The eye was proptosed. An incision about six to eight mm. from the limbus was made in the conjunctiva which was dissected free of the sclera. Under direct vision, a suture of any of the materials already mentioned was passed through the sclera into the vitreous cavity and cut and tied. The conjunctiva was closed by a 6-0 catgut running suture.

The eyes were examined from time to time. The eyes were enucleated after 48 hours, one week, two weeks, four weeks, and eight weeks.

In each experimental evaluation a minimum of three animals were employed.

Experimental results

Forty-eight hours

Plain gut 5-0, Five animals, 10 eyes

Ophthalmoscopic examination. The anterior media were clear, vitreous showed some floaters, and occasional spotlike hemorrhage in one case. In the same case the retina around the suture was edematous and flame-shaped hemorrhages around the suture area were seen.

* From the Research Department of the Wills Eye Hospital. This study was supported in part by Training Grant 2B-5076C(2). Presented at the IX Annual Clinical Conference of Wills Eye Hospital, February, 1957.

Fig. 1 (Paul and Fry). Plain gut suture after 48 hours. Microphotograph of the suture area, showing: (A) section through the suture, (B) subretinal hemorrhage, little or no cellular reaction.

Gross examination (macroscopic). Out of 10 eyes enucleated only one showed slight detachment, with edema and hemorrhage around the suture point. The rest of the eyes looked normal.

Microscopic examination (fig. 1). The angle of the anterior chamber was free of adhesions. Vessels were normal. There was slight round-cell infiltration, with complete absence of acute inflammatory cells. Iris and ciliary body stroma appeared healthy.

In the retina surrounding the suture area, fresh subretinal hemorrhage was noticed in one eye. The retina overlying the area appeared healthy. Little or no cellular reaction was present. The retina in the rest of the eye looked healthy.

Chromic gut 6-0, five animals

Ophthalmoscopic examination. The anterior media were clear. Vitreous floaters and opacities could be seen. There were flame-shaped and punctate hemorrhages around the suture area in four eyes. Edema of the retina was present at the suture point in four eyes. Detachment of the retina could be seen in one eye.

Macroscopic. Three eyes showed slight detachment around the suture point. One eye showed extensive detachment. Hemorrhage in the retina and vitreous was seen in one eye; hemorrhage in the retina in four eyes (all). The rest of the eyes appeared normal.

Microscopic. The angle of the anterior chamber was open with no adhesions. The vessels looked normal. There was no cellular infiltration at the angle or in the iris and ciliary body stroma. The iris and ciliary body stroma looked normal (fig. 2-A).

Retina surrounding the suture area. At and a little around the suture, disruption of the retinal structure was seen. There was no cellular reaction

Fig. 2-A (Paul and Fry). Chromic gut suture after 48 hours. Microphotograph showing the angle of the anterior chamber. No adhesions and an open angle, no evidence of inflammation of the iris.

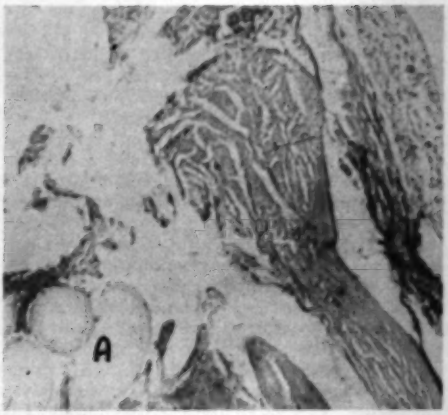

Fig. 2-B (Paul and Fry). Chromic gut suture after 48 hours. Microphotograph through the area of the suture. (A) Little or no cellular reaction in the retina.

or infiltration. The retina of the rest of the eye looked normal (fig. 2-B).

Nylon 6-0, five animals

Ophthalmoscopic examination. The anterior media were clear. Vitreous opacities and isolated punctate hemorrhages were seen in two eyes. No visible abnormality was seen in the vitreous of the other eyes.

Macroscopic. Grossly the eyes looked normal. No exudation, hemorrhage, or detachment were present. One eye out of the total number showed slight hemorrhage at the suture point.

Microscopic. The angle of the anterior chamber was free of adhesions. An aggregation of round cells and pigment cells was seen. Occasional polymorphonuclear cells also were seen. The iris stroma showed dispersal of pigment cells in its entirety; occasional round cell infiltration was seen. This was also true of the ciliary body stroma. Vascular engorgement was also noted.

Around the suture. Cellular infiltration of the sclera and choroid was present. The cells were mainly round cells and acute inflammatory cells. There were spotted hemorrhages around the suture track. In the immediate vicinity of the suture track, there was destruction of retinal structure followed by cystic degeneration for some distance. The rest of the retina looked healthy.

Silk 6-0, five animals

Ophthalmoscopic examination. The anterior media were clear. Vitreous hemorrhage was seen in three eyes. No visible abnormality was seen in the rest of the eyes. An area of chorioretinitis was present in the region of the suture.

Macroscopic. Grossly the eyes looked normal. Small, flamed-shaped retinal hemorrhage was noted in three eyes. No retinal detachment was seen in any one of the eyes.

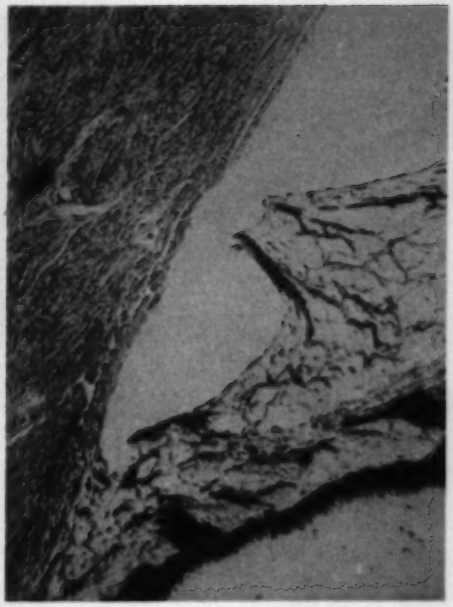

Fig. 3-A (Paul and Fry). Silk suture after 48 hours. Microphotograph showing an open angle with no evidence of inflammation in the iris.

Microscopic. The angle of the anterior chamber was open. No adhesions were seen. Iris and ciliary body stroma appeared normal. No cellular infiltration was seen (fig. 3-A).

Around the suture. The sclera appeared normal (fig. 3-B). No cellular infiltration was present.

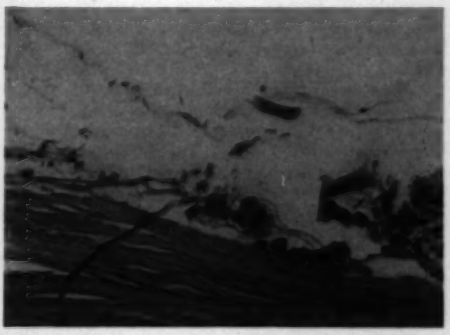

Fig. 3-B (Paul and Fry). Microphotograph of silk suture through the retina after 48 hours. Disruption of the retinal architecture with early evidence of gliosis of the neighboring retina.

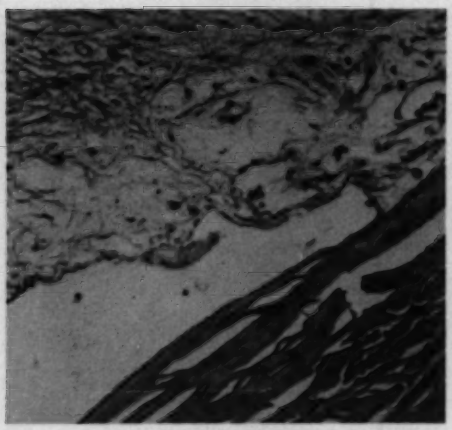

Fig. 4-A (Paul and Fry). Microphotograph of the angle of the anterior chamber of the eye with chromic gut suture after one week. Note the open angle with no cellular infiltration or adhesion.

The choroid in the region of the overlying suture was thickened. Choroid and retina were firmly adherent in the region of the suture. Retinal structure was disrupted in that area. No fibrosis was seen. There was some evidence of early gliosis. The rest of the retina looked normal.

ONE WEEK

Plain gut, 5-0

Ophthalmoscopic examination. The media were fairly clear but for stray vitreous floaters. The retina looked normal.

Macroscopic examination. On gross examination no pathologic alteration was detected. No exudation, hemorrhage, or retinal detachment were present.

Microscopic examination. Examination of the angle of the anterior chamber showed plasma-cell infiltration of the stroma of the iris near the angle. The angle was free of adhesions. The ciliary body was slightly infiltrated with plasma cells. The rest of the stroma of the iris looked normal.

Around the suture area. The sclera appeared normal. Near the suture track the retinal structure had degenerated for some distance. The suture track in the retina showed round-cell infiltration. The sclera in the suture track showed chronic inflammatory cells. The rest of the retina looked normal. The outer coats of the eye showed no widespread inflammatory reaction.

Chromic gut, 6-0

External eye examination indicated no abnormality.

Ophthalmoscopic examination. In one eye there was edema and slight detachment of the retina around the suture area. Vitreous floaters were present. Another eye showed hemorrhage in the vitreous.

Macroscopic. Two eyes showed slight detachment of the retina at the suture point. The vitreous was healthy in all cases. In one rabbit retinal hemorrhage was seen at the suture point.

Microscopic. The angle of anterior chamber was open. No adhesions were present, and no cellular infiltrates. The stroma of the iris and ciliary body looked normal. No vascular congestion could be seen (fig. 4-A).

Around the suture area (fig. 4-B). There was destruction of the retinal architecture around the suture track. No cellular infiltrates were seen around the track. One fresh hemorrhage was seen in the retina in one eye.

Silk, 6-0

Ophthalmoscopic examination. The media were fairly clear. Occasional floaters were noted. The eyes looked quiet. No detachment or vitreous hemorrhages could be seen.

Macroscopic. No gross abnormality was seen and the vitreous looked healthy. No retinal detachment or hemorrhages were present.

Fig. 4-B (Paul and Fry). Microphotograph of chromic gut suture through the retina (A) after one week, showing destruction of the retinal architecture in the immediate vicinity of the suture. Note the cellular infiltrates in the gap and fibrous tissue proliferation.

Microscopic. The angle of the anterior chamber was open. There were no adhesions or exudations and no cellular infiltrates. Iris and ciliary body stroma looked normal (fig. 5-A).

Around the suture area. Cystic degeneration of the retina appeared around the suture track. There was destruction of the retinal structure for some distance around the track, with proliferation of pigment epithelium in that area. The choroid was thickened. No cellular reaction could be seen. The rest of the retina looked normal (fig. 5-B).

Nylon, 6-0

Ophthalmoscopic. The media were slightly hazy. Vitreous opacities and floaters were present. No retinal detachment or hemorrhages were seen.

Macroscopic. The vitreous looked fluid. No retinal detachment or hemorrhages were seen.

Microscopic. The angle of the anterior chamber was closed by fibrous tissue and chronic inflammatory cell infiltration. Iris and ciliary body were congested, with chronic inflammatory cell infiltration. Vascular engorgement was present, as were a few anterior peripheral and posterior synechias.

Around the suture (fig. 6). Sclera: Chronic inflammatory granuloma was seen around the nylon suture, surrounded by blood elements, polymorphonuclear cells, lymphocytes, necrotic tissue, and fibrous tissue. This fibrous mass traversed the suture track up to the retina.

Retina and choroid. The choroid was thickened, with engorgement of blood vessels and infiltration of polymorphonuclear leukocytes and various round cells. The retinal structure was destroyed around the suture track. There was infiltration of the retina by polymorphonuclear leukocytes and round cells and fibrous tissue encapsulation around the suture. The rest of the retina looked normal. Some distance away from the suture track the retina looked normal.

Fig. 5-A (Paul and Fry). Microphotograph of the angle in an eye with silk suture after one week. Note the open angle with no adhesion and no inflammatory reaction.

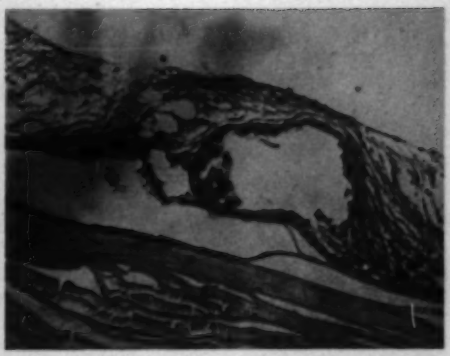

Fig. 5-B (Paul and Fry). Microphotograph of silk suture after one week, showing cystic degeneration in the retina around the suture track and early gliosis.

TWO WEEKS

Plain gut, 5-0

Ophthalmoscopic examination. The media were clear. A patch of chorioretinitic atrophy was seen at the suture point. No other abnormality was seen. No hemorrhages or retinal detachment were seen.

Macroscopic. The vitreous looked normal. No hemorrhage or detachment of the retina was present. Grossly the eyes looked normal.

Microscopic. The angle of the anterior chamber was open. No synechias were seen. There was no cellular infiltration except for an occasional polymorphonuclear leukocyte and lymphocyte. Iris and ciliary body stroma appeared normal. There was no vascular engorgement or exudation (fig. 7-A).

Around the suture. Sclera: Polymorphonuclear leukocytic reactions were seen around the suture and the track. The suture was encapsulated by fibrous tissue. Retina and choroid: Fibroblastic reaction was present in the suture track. There was more plasma-cell infiltration than polymorphonuclear. Thickening of the choroid was seen in the area of the track. There was destruction of retinal structure for some distance around the suture and a slight attempt at gliosis. The rest of the retina looked normal (fig. 7-B).

Chromic gut, 6-0

Ophthalmoscopic examination. The media were clear. No vitreous opacities were seen. The fundus looked normal. No hemorrhage or retinal detachment were present.

Macroscopic. All eyes looked normal grossly.

Fig. 6 (Paul and Fry). Microphotograph of the suture track of a nylon suture after one week, showing chronic inflammatory cells and fibrous tissue in and around the suture track.

The vitreous looked normal. No hemorrhage or retinal detachment were seen.

Microscopic. The angle of the anterior chamber was open. No adhesions were present at the angle. There were no cellular infiltrations in the angle, the iris, or the ciliary body. The stroma of the iris and ciliary body appeared normal.

Around the suture area. Sclera: There was chronic inflammatory reaction with fibroplasia. A fibrous band infiltrated with round cells and occasional polymorphonuclear leukocytes was seen traversing the suture track and reaching up to the retina. Choroid and retina: Proliferation and migration of the choroidal and retinal pigment cells both in the outer and inner coats of the eye could be seen. The retinal structure was destroyed around the suture track, with occasional clusters of round cells and stray polymorphonuclear leukocytes in the retina at that area. The fibrous band traversing the suture track spread out on the retina. Gliosis of the retina was seen for some distance. The rest of the retina looked normal (fig. 8).

Silk, 6-0

Ophthalmoscopic examination. The media were clear and there was no vitreous haze. The retina looked normal. No hemorrhage or retinal detachment could be seen.

Macroscopic. The anterior segment looked normal, as did the vitreous. No retinal detachment or hemorrhages were seen.

Microscopic. The angle of the anterior chamber

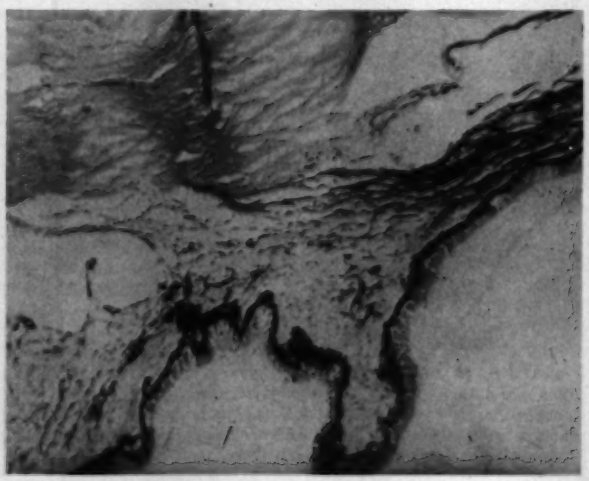

Fig. 7-A (Paul and Fry). Microphotograph of angle of the anterior chamber in an eye with plain gut sutures after two weeks. Note the open angle with no adhesion.

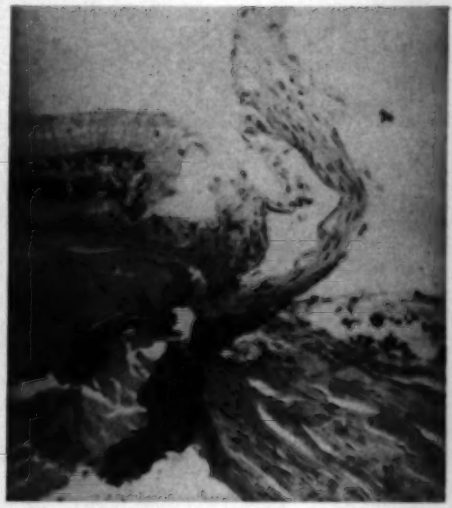

Fig. 7-B (Paul and Fry). Microphotograph of the suture track of plain gut suture after two weeks, showing chronic inflammatory cellular reaction, and fibrous tissue proliferation.

was open. No adhesions were present and no cellular infiltration. The stroma of the iris and ciliary body appeared normal.

Around the suture. Sclera: Slight infiltration of the suture track in the sclera by polymorphonuclear leukocytes and round cells could be seen. A fibrous structure filled the track and extended into the retina. The choroid looked normal. The retinal structure was destroyed for some distance around the suture track. No cellular infiltration of the retina could be seen, although there was some attempt at gliosis. The rest of the retina appeared normal (fig. 9).

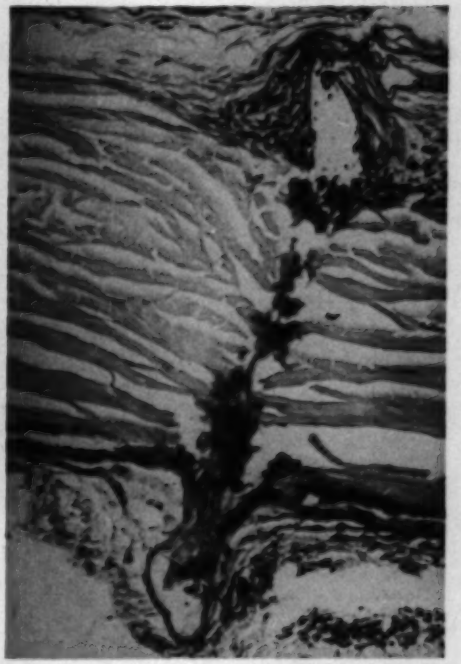

Fig. 8 (Paul and Fry). Microphotograph through the suture track of chromic gut suture after two weeks, showing proliferation of pigment cells, fibrous tissue proliferation, and gliosis of the retina.

Nylon, 6-0

Ophthalmoscopic examination. The media were hazy. The fundus looked normal. No detachment or hemorrhage were seen in the retina. The anterior segment appeared normal.

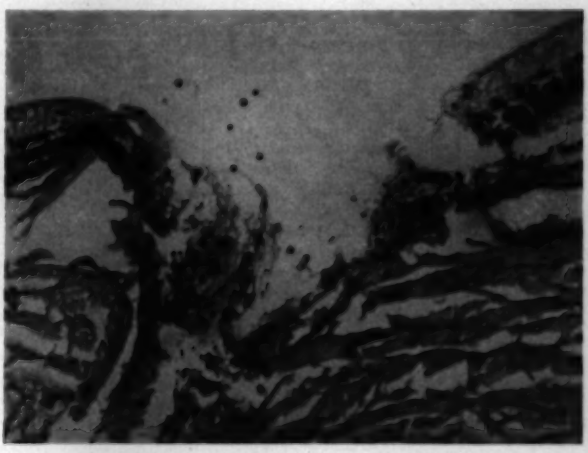

Fig. 9 (Paul and Fry). Silk suture in the retina after two weeks. Microphotograph, showing round-cell infiltration of the suture track and slight gliosis of the retina.

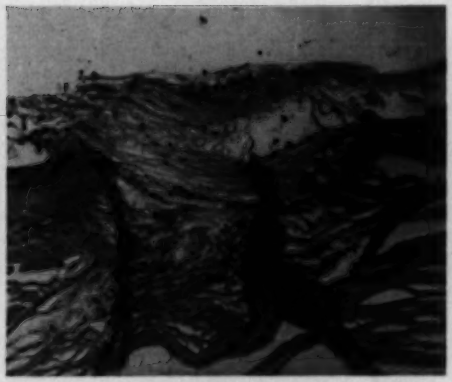

Fig. 10-A (Paul and Fry). Plain gut four weeks. Microphotograph of the suture track, showing round-cell and plasma-cell reaction with gliosis. Note the destruction of retinal architecture.

Macroscopic. The vitreous was cloudy; the retina normal. The anterior segment looked normal.

Microscopic. The angle of anterior chamber was open. No adhesions were apparent. There was no cellular infiltration either in the angle or the stroma of the ciliary body or iris. The iris and ciliary body looked normal.

Around the suture. Sclera: There was encapsulation of the suture by fibrous tissue. Cellular infiltration showed mainly epithelial cells, round cells, and occasional polymorphonuclear leukocytes. The choroid was slightly thickened. No cellular infiltration was present. Retina: The retinal structure was destroyed around the suture track. No cellular infiltration could be seen but there was a slight attempt at gliosis. The rest of the retina looked normal.

FOUR WEEKS

Plain gut, 5-0

Ophthalmoscopic examination. The media were clear. No abnormality was seen in the fundus.

Macroscopic. The anterior segment looked normal, as did the vitreous. No hemorrhage or retinal detachment could be seen.

Microscopic. The angle of the anterior chamber was open. No adhesions were present. No cellular infiltration could be seen either at the angle or of the stroma of the iris and ciliary body which appeared normal.

Around the suture. Sclera: There was round-cell and epithelioid-cell reaction in the suture track, and fibrous tissue reaction in the suture track. A mass of undifferentiated cells filled up the tangle of fibrous tissue meshwork in the suture track which extended to the retina. Choroid and retina: The choroid looked normal. The retinal structure was destroyed around the suture track. There was extensive gliosis in the neighboring retina, with round-cell infiltration. The rest of the retina was normal (fig. 10-A).

Silk, 6-0

Ophthalmoscopic examination. The media were clear. No retinal detachment, hemorrhage, or inflammation could be seen.

Macroscopic. The anterior segment looked normal. The vitreous was clear. No detachment or retinal hemorrhage could be seen.

Microscopic. The angle of anterior chamber was open, with no adhesions. No cellular infiltration of either the angle or the stroma of the iris and ciliary body was apparent. The stroma of the iris and ciliary body appeared normal (fig. 11-A).

Around the suture. Sclera: There were permeation of the suture fibers by fibrous tissue and encapsulation of the whole silk suture by fibrous tissue and infiltration in and around by epithelioid cells, spindle cells, and round cells. Solitary occasional polymorphonuclear cells could be visualized. Retina: Suture fibers were permeated by fibrous tissue, with encapsulation of the whole bundle of silk suture. The overlying retinal structure was disrupted. Evidence of overlying and neighboring retinal gliosis was present. The choroid was slightly thickened. No cellular infiltration could be seen. The rest of the retina was normal (fig. 11-B).

Nylon, 6-0

Ophthalmoscopic examination. The media were fairly clear. The vitreous appeared fluid and hazy. There were atrophic changes in the retina at the suture point. No hemorrhage or retinal detachment could be seen.

Macroscopic. The anterior segment looked nor-

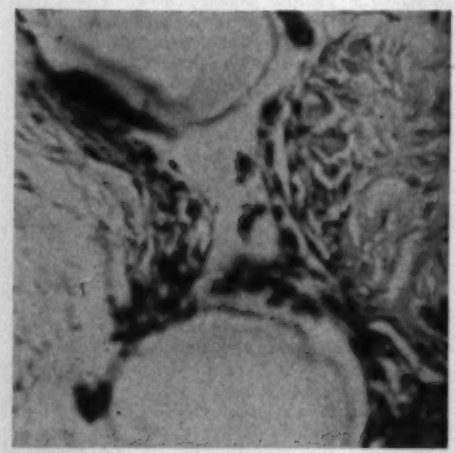

Fig. 10-B (Paul and Fry). Nylon after four weeks. Microphotograph of the suture, showing extensive fibrous tissue reaction and inflammatory cellular infiltration.

mal. The vitreous was fluid with strands. The retina looked atrophic at the suture point.

Microscopic. The angle of the anterior chamber showed no adhesions and was open. There was some infiltration of the root of the iris by round cells. Iris and ciliary body stroma seemed relatively free of infiltration.

Around the suture. Sclera: Extensive fibrous tissue reaction could be seen around the nylon suture, resulting in its encapsulation. Fibroblastic, epithelioid cells, plasma cell, and occasional polymorphonuclear infiltration were seen. Pigment-cell scattering in that area was noticed. Choroid and retina: The choroid had disappeared at the site of the suture and was thickened around the suture. There was destruction of the retinal structure at and around the suture track. Generalized infiltration was present with epithelioid and plasma cells and fibroblastic reaction. Hemorrhagic spots could be seen in the vitreous. There were occasional attempts at gliosis and cystic degeneration. The rest of the retina looked normal (fig. 10-B).

EIGHT WEEKS

Chromic gut, 6-0

Ophthalmoscopic examination. The media were clear. No abnormality was seen in the fundus.

Macroscopic. The anterior segment looked normal, as did the vitreous. No hemorrhage or retinal detachment could be seen.

Microscopic. The angle of the anterior chamber showed slight adhesions by fibrous tissue and occasional round and pigment cell infiltration. Iris and ciliary body stroma were normal (fig. 12-A).

Around the suture. Sclera: Fresh hemorrhage from newly formed blood vessels was seen, as were pigment cell dispersal and occasional round cells.

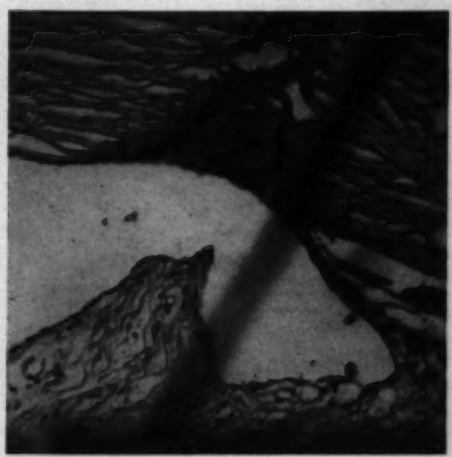

Fig. 11-A (Paul and Fry). Silk after four weeks. Microphotograph, showing an open angle and no evidence of inflammatory reaction.

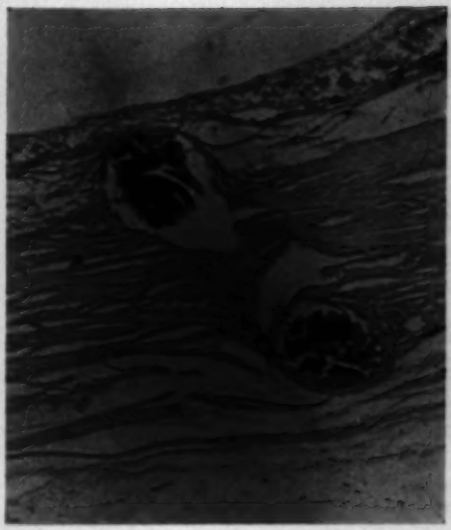

Fig. 11-B (Paul and Fry). Silk after four weeks. Microphotograph of the suture track, showing gliosis of the neighboring retina and no cellular infiltration.

Choroid and retina: The choroid was disintegrated. A granulomatous nodule at the suture site was composed of a loose areolar network, enmeshed in which were occasional polymorphonuclear leukocytes, plasma cells, spindle-shaped fibroblasts, and epithelioid cells. Dispersal of pigment in that area was seen. The rest of the retina looked normal (fig. 12-B).

Silk, 6-0

Ophthalmoscopic examination. The media were clear. No abnormality could be detected in the fundus.

Macroscopic. The anterior segment looked normal. The vitreous was normal. No hemorrhage or retinal detachment could be seen.

Microscopic. The anterior chamber angle was open and there were no adhesions. No cellular infiltrates were seen either in the angle, the stroma of the iris, or the ciliary body. The iris and ciliary body were normal (fig. 13-A).

Around the suture area. Sclera: Encapsulation and permeation by fibrous tissue of the silk suture were noted. There were few foreign body giant cells, round cells, plasma cells, and occasional lymphocytes. Choroid and retina: The choroid had disappeared over the area. No cellular infiltrates were seen. There was complete destruction of the structure of the overlying retina, which appeared like a fibrous tissue sheet. No cellular infiltration was detected. Gliosis of the neighboring retina was apparent. There were encapsulation and permeation by fibrous tissue of the silk suture. The rest of the retina looked normal (fig. 13-B).

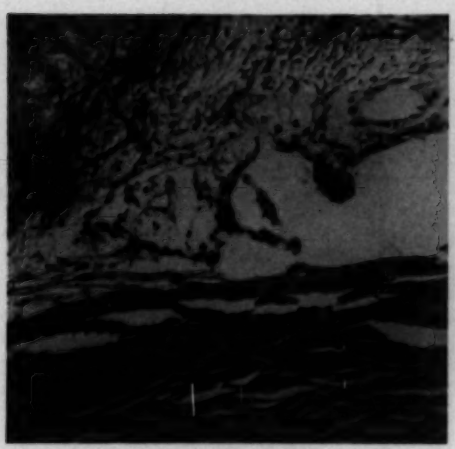

Fig. 12-A (Paul and Fry). Chromic gut after eight weeks. Microphotograph, showing adhesion at the angle.

Nylon, 6-0

Microscopic. The angle of anterior chamber was open. No adhesions and no cellular infiltration of the angle, the iris, and the ciliary body were present. The iris and ciliary body stroma appeared normal (fig. 14-A).

Around the suture. Sclera: There was extensive fibrotic reaction, with infiltration by round cells, epithelioid cells, fibroblasts, plasma cells, and occasional polymorphonuclear leukocytes. It looked like a granuloma. This granuloma traversed the suture track down to the retina. Choroid and retina: Complete disappearance of choroid in the neighboring area was noted, with dispersal of pigment and destruction of retinal structure. A granulomatous nodulelike mass protruded through the suture track into the vitreous cavity. It was comprised of fibrous tissue, plasma cells, and round cells. Cellular infiltration of the neighboring retina was also seen. Gliosis and cystic degeneration of the neighboring retina were seen. The rest of the retina appeared normal (fig. 14-B).

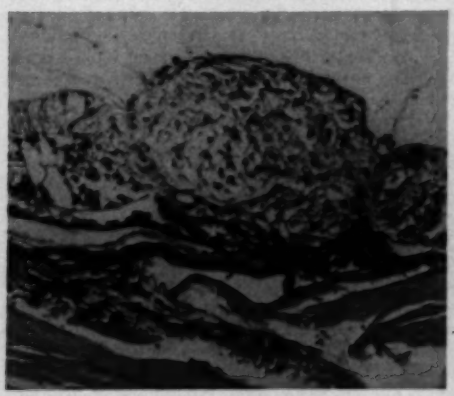

Fig. 12-B (Paul and Fry). Chromic gut after eight weeks. Microphotograph, showing granulomatous nodule at the suture site composed of fibrous network and plasma cells.

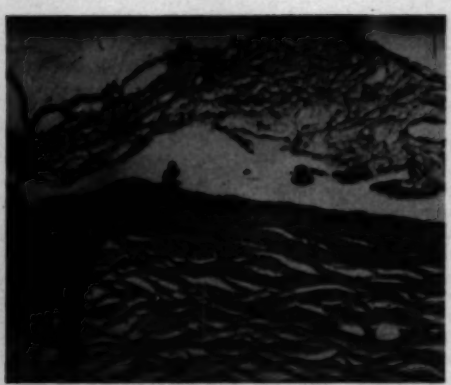

Fig. 13-A (Paul and Fry). Silk after eight weeks. The angle of the anterior chamber was open, with no adhesion or evidence of inflammatory reaction.

Fig. 13-B (Paul and Fry). Silk after eight weeks. Microphotograph through the suture (A) track, showing extensive gliosis of the retina.

Fig. 14-A (Paul and Fry). Nylon after eight weeks. Microphotograph of the angle of the anterior chamber which is open, with no adhesion.

Comment and Summary

It is now established that the actual treatment of detachment of the retina (by a surgical procedure) consists in creating at a given place inflammatory adhesions between the retina and the choroid. The agents capable of producing inflammations are numerous.

It is imperative that the manner of production of inflammation should be such that the proliferative factor is dominant over the exudative factor. It is penetration by the proliferative cells of the choroid and of the retina that produces a firm adhesion which is solid and durable. Moreover, this provoked inflammation should be so regulated that it is not so severe as to create a marked reaction in the choroid and retina. In the third place it is necessary to fix the retina to the choroid at the point of application of the irritating agent. Last but not the least, the procedure must be capable of easy execution.

It is well known that the first condition (proliferation dominating exudation) is easily satisfied by mechanical irritation. During our studies it was confirmed that sutures, in addition to satisfying the first condition, also satisfy the other three.

Forty-eight hours after applying a suture to the retina, plain gut caused subretinal and retinal hemorrhages but no reaction. Chromic gut did not produce any adverse reaction. Silk caused thickening of the choroid and no cellular reaction. Nylon caused a severe acute inflammatory reaction.

After one week, plain gut produced a mild iridocyclitis and a chronic inflammatory reaction around the suture track. Chromic gut produced retinal hemorrhages but no cellular infiltration. Silk produced thickening of the choroid without any cellular infiltration. Nylon, however, produced a severe reaction; all coats of the eye were affected.

Two weeks. With plain gut, the iridocyclitis had disappeared but in the sclera and inner coats of the eye an inflammatory reaction remained. With chromic gut the reaction was about the same as with plain gut but there was more choroiditis. With silk the reaction was very mild as compared to the reactions of chromic gut and plain gut. Nylon, however, still produced the severest reaction of all.

Four weeks. With plain gut, the inflammation had subsided considerably but there was evidence of fibrosis and round-cell infiltration. With silk the reaction was about the same as with plain gut. Gliosis and fibrosis

Fig. 14-B (Paul and Fry). Nylon after eight weeks. Microphotograph of the suture track, showing a granulomatouslike mass protruding into the vitreous cavity and comprised of fibrous tissue, polymorphonuclear leukocytes, and plasma cells.

were more apparent than with plain gut. After four weeks, nylon produced a chronic iridocyclitis and slight degeneration of the vitreous. The sclera, choroid, and retina exhibited chronic inflammation, with fibrosis in the choroid and retina accompanied by cystic degeneration of the retina and also by gliosis.

Eight weeks. With plain gut, absorption has proceeded to a point where the area of insertion could not be identified. With chromic gut the inflammatory reaction produced a partial closure of the angle of the anterior chamber. A few round-cell infiltrates in the iris and ciliary body were noted. Chronic inflammatory cells were found in the inner coats of the eye, with extensive fibroblastic reaction. Silk produced little or no reaction in the inner coats of the eye except for some fibroblastic reaction and gliosis. There was no evidence of uveitis. Nylon produced a foreign body granulomatous reaction accompanied by cystic degeneration of the retina. The uvea seemed to be relatively free of inflammation.

Conclusion

From this study the following conclusions may be made:

1. Silk caused no undesirable inflammatory reaction in the eye.
2. Chromic gut was not tolerated quite as well as silk.
3. Nylon produced a severe reaction.
4. The reaction produced by silk or chromic gut was confined to the area of the suture. It did not affect the rest of the retina which appeared normal.
5. Plain gut produced an initial acute inflammatory reaction and iridocyclitis. After absorption this reaction subsided.
6. With all materials, two months after insertion of the sutures, the retina at and in the immediate neighborhood of the sutures remained in firm contact with the choroid and the sclera.

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Acknowledgment

We wish to acknowledge the encouragement and helpful discussions by Dr. Irving H. Leopold, Director of Research, during the course of this investigation.

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NOTES, CASES, INSTRUMENTS

LID RETRACTION SYNDROME*

Due to "secondary deviation"

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Pueblo, Colorado

Retraction of the upper lid with widening of the palpebral fissure may be a symptom or sign of one of several pathologic conditions. Except for the cases associated with thyroid disease it is a fairly rare condition. It may conveniently be divided into two types: (1) spasmodic or intermittent, and (2) constant.

Spasmodic retraction of the upper lid has been described in exophthalmic goiter, irritative lesions of the sympathetic chain, and dental lesions (reflex sympathetic stimulation). It has also been attributed to syphilitic involvement of the cerebral nuclei. With the exception of sympathetic stimulation to smooth muscle, causative factors have not been well understood. According to Walsh,[1] elevation of the upper lid associated with movement of the globe is sometimes observed as a result of misdirection of regenerated third-nerve fibers. The pseudo von Graefe's sign, in which the lid fails to follow the globe downward, is a result of this situation. Walsh states that abnormal retraction of the lid is sometimes seen in myasthenia gravis.

The condition termed cyclic oculomotor palsy exhibits alternate retraction and ptosis of the upper lid but there are associated movements of the globe and usually pupillary abnormalities. A case of periodic elevation of the upper lid as a result of clonic spasm of the levator was reported by Posey in an eight-year-old boy, with no associated movement of the globe. He considered this case to be due to chorea.

Sustained retraction of the upper lid is an outstanding sign of toxic goiter. It usually is bilateral but may be unilateral. Stellwag's sign (retraction and infrequent blinking), Dalrymple's sign (abnormal widening of the palpebral fissure), and von Graefe's sign (lid lag) are all due to a tonic contraction of the smooth muscle in the upper lid (Müller's muscle).

Sustained retraction has been described in epidemic encephalitis, tumors of the midbrain, bulbar polio, hydrocephalus, and in several cases where the etiology could not be determined. Walsh states that he has observed two or three such cases of retraction of one or both upper lids where all examinations other than of the eyelids were negative. In the cases he observed, the symptoms persisted for a few days or weeks and then disappeared spontaneously.

Case report

B. G., a 48-year-old Negress, was admitted to the orthopedic service of Jefferson Davis Hospital on July 19, 1954, following an auto accident in which she sustained multiple fractures of the lower extremities with abrasions and contusions of the face and a fracture dislocation of the right ulna. The ophthalmology service was called to see the patient because of conjunctival hemorrhage and ecchymosis around the right eye. The patient stated that the right eye was her better one and that vision in the left eye had been poor since the age of 18 or 20 years.

Examination revealed a large hematoma of the right upper lid with resulting ptosis. The left eye was uninjured. Vision was grossly equal in each eye. There was no hyphema. Pupils were dilated with 10 percent Neosynephrine; both dilated well. The fundus was normal in the right eye; in the left there was a central posterior polar cataract, otherwise normal. X-ray films of the orbit were negative for fracture. Ophthalmologic diagnosis was contusion injury right eye with ptosis and posterior polar cataract left eye. The patient was treated with continuous hot, moist compresses to the right eye.

On July 25th, the eye service was asked to see the patient again because of the development of "exophthalmos" or "proptosis" of the left eye with lid lag. At this time there was a notable widening of the palpebral fissure on the left which had not been present on July 19th. There was moderate lag on downward gaze. There was still edema and swelling of the right upper lid so that there was

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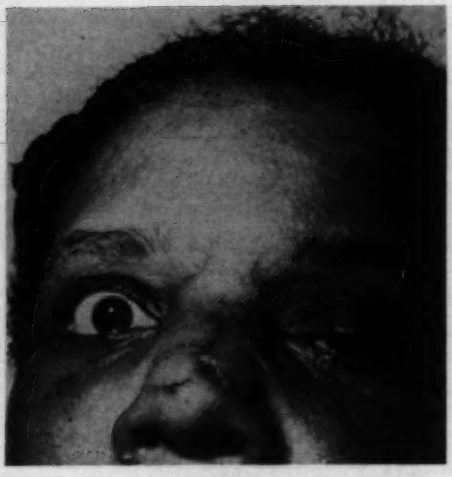

Fig. 1 (Lewallen). Sustained retraction of the left upper lid, with traumatic ptosis of the right lid.

a relative and a partially real ptosis on the right. Conjugate movements were normal with no involvement of any extraocular muscle. There was no diplopia in any field. The near-point of convergence was recessed to 180 mm.

At this point the widening of the fissure was thought to be due to or related to trauma to the cervical spine and it was felt that the ptosis on the right merely masked the symptom on that side. X-rays studies of the cervical and thoracic spine were negative and neurologic examination was negative. Hertel exophthalmometer readings were equal on the two sides. On August 4th, the ptosis of the right eye was more marked and with the frontalis immobolized she was unable to elevate the lid. The palpebral fissures measured 5.0 mm., O.D., and 11 mm., O.S. Horner's test showed no response to adrenalin but four-percent cocaine to the left eye produced dilation of the pupil and an increase in the lid retraction. Serology was negative.

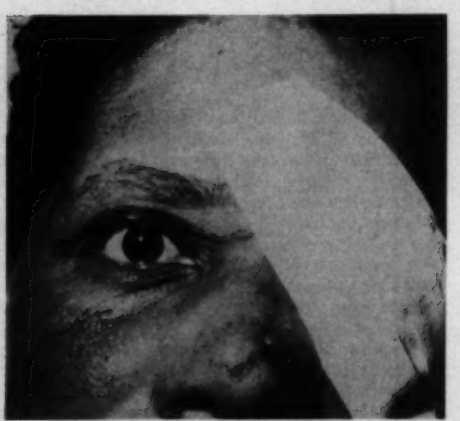

Fig. 2 (Lewallen). Same patient following patching of the right eye for 48 hours, proving the "secondary deviation" etiology.

At this point the thought occurred that the levators could be acting as "yolk" muscles according to Herring's law and that, with poor vision in the left eye, the patient was making a great effort to raise the ptosed right lid, thus providing a secondary deviation of the left upper lid.

In an attempt to prove this idea the ptosed right eye was patched tightly and the left eye carefully observed for a change in appearance. Within 48 hours the left lid came down to a normal position, measuring 8.5 mm. On removal of the patch, the left lid again assumed a higher position after 24 hours thus proving the theory of secondary deviation as the etiologic factor.

The ptosis of the right lid cleared gradually; on October 18th vision was found to be 20/50, O.D., and 20/100, O.S., and on November 23rd, the fissures were equal at 9.0 mm.

Discussion

Widening of the palpebral fissure is apparently quite rare when cases of toxic goiter are excluded. As a matter of fact Fuchs' *Diseases of the Eye* (1933)[2] states that spasms of the levator do not occur except in a form of secondary contraction from paralysis of the orbicularis. It is interesting to note that this same edition of Fuchs' textbook contains a picture of a patient with ptosis of the left lid and widening of the opposite fissure. The explanation given is that the patient elevated the brow on either side and thus raised the right lid higher than the left. This patient also had poor vision in the uninvolved eye.

The development of the symptom in the case herein reported seems to be contingent on several factors: ptosis of the one eye when vision in the opposite eye is sufficiently depressed so that the patient makes a great effort to use the good eye with the ptosis. It is very doubtful that this symptom would develop in a situation where the vision was equal in both eyes, even though the ptosed eye might be the dominant one.

The levators are not commonly thought of

as extraocular muscles and no mention was found in any of the textbooks consulted of the application of Herring's law to them. However, it would seem from this case that they do function as "yolk muscles."

105 East Pitkin.

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BOECK'S SARCOID OF THE ORBIT*

REPORT OF A CASE RECEIVING QUESTIONABLE BENEFIT FROM STEROID THERAPY

WILLIAM A. SMITH, M.D.
Atlanta, Georgia

It has been well known for many years that the eyes and the ocular adnexa are frequently involved with generalized sarcoidosis. Freiman, in 1948, stated that the eye may be involved in 25 to 50 percent of cases of sarcoidosis. As enumerated by Levitt, structures in and about the eye which may be affected include the skin of the lids, the conjunctiva, the lacrimal gland, the episclera, the cornea, the uveal tract, the retina, and the optic nerve. Up to 1941, however, no authentic cases of Boeck's sarcoid of the orbit had been reported except one by King, in 1939.

Since Levitt's review of ocular sarcoidosis, there have been eight more cases of orbital involvement proven by biopsy and supportive findings reported in the literature. In the majority of these cases a palpable tumor was felt through the lid prior to surgery. Benedict, in 1949, reviewed 1,000 pathologic slides of orbital tumors at the Mayo Clinic and found only two cases of Boeck's sarcoid of the orbit, the surgical procedures having been done in 1926 and 1948.

The case to be presented, which makes the 10th reported, is the first to be treated with long-term steroid therapy. The result of this prolonged treatment is equivocal since sarcoidosis is known to regress spontaneously at times without any treatment.

* From Harper Hospital, Detroit, Michigan.

Of the orbital cases reported previously, one eye had to be removed because of phthisis bulbi. In one case the levator palpebra muscle, the superior rectus muscle, and the lateral rectus muscle were destroyed in removing the affected tissue. In another case the lacrimal gland and the lateral rectus had to be excised. In a fourth case the patient continued to have proptosis, ptosis, and diplopia despite surgical and X-ray therapy. Thus, any new treatment considered of value should be tried on these patients.

CASE REPORT

A 65-year-old Negress, Mrs. A. C., was seen in the Harper Hospital eye clinic on June 25, 1956, because of pain in her left eye that was quite severe. She had been told by a relative one month previously that her left eye appeared more prominent than her right eye. Two weeks prior to being seen she began having pain in her left eye and also in the right parietal and occipital regions. The pain had become progressively more severe. The day after the pain started, her left upper lid began to droop. During this previous month her left eye became more and more prominent.

On examination, the patient was found to be an obese, well-nourished individual who was obviously in severe distress due to the marked pain in her head. Vision was 20/40, O.U., uncorrected; ocular tension was 22 mm. Hg (Schiøtz), O.U.; exophthalmometer readings were 24 mm., O.D., and 26 mm., O.S., with a base measurement of 98 mm. She had almost complete loss of the fourth, sixth, and third nerves on the left side except for pupillary reaction to light. Her visual fields were normal.

She was admitted to the hospital on the same day with a diagnosis of possible arteriovenous aneurysm of the internal carotid artery. This was based on the acuteness of her symptoms, although very little vascular engorgement was evident and no bruit was heard in her head.

Laboratory work disclosed a normal urine with specific gravity of 1.018, a trace of albumin, and no sugar. She had a CBC with 4.40 RBC, and 8,600 WBC. She had a FBS of III, a negative

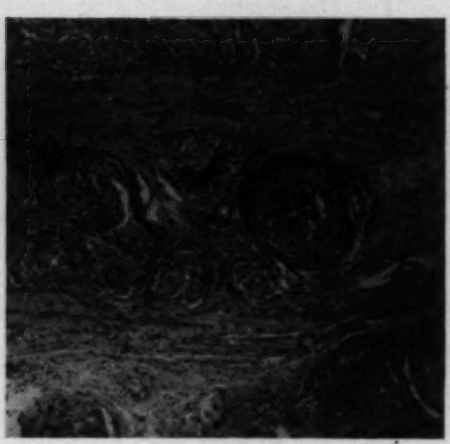

Fig. 1 (Smith). Microscopic appearance of biopsied tissue.

Kahn, a total protein of 6.3 gm. with 4.4 gm. of albumin, 1.8 gm. of globulin, and a A/G ratio of 2.4. Her sedimentation rate was 46.

On June 26, 1956, 100 mc. of radioactive phosphorus (P^{32}) were given intravenously and 24 hours later definite localization of a tumor mass was made in the posterior portion of the left orbit. This was confirmed on June 29th, when a repeat count was made with a scintillating counter and a Geiger counter. At this time a questionable mass was felt in the superior portion of the left orbit through the upper lid. On July 6th, an arteriogram was performed and was negative. On July 10th, a lateral orbitotomy of the left orbit was done, using a modified Krönlein procedure. Only a small mass was found in the posterior part of the orbit just medial to the optic nerve. A biopsy showed microscopically lesions typical of sarcoidosis within muscle tissue (fig. 1). It was therefore presumed that the tumor arose from the medial rectus muscle.

Following the operation further studies disclosed a negative skin reaction to old tuberculin (1:100,000). An X-ray film of her chest was normal, but X-ray films of her hands showed a well-demarcated area of rarefaction measuring 5.0 by 10 mm. in the distal shaft of the proximal phalanx of her left thumb, which was suggestive of sarcoidosis.

On July 17th, the patient was started on 50 units of ACTH twice daily which was gradually reduced over a 10-day period. She was discharged on July 26th with Meticorten (5.0 mg.) to take at home three times daily. The condition of her left eye was very little improved except that the pain was much less and she could move her eye a little toward the midline. However, she could only raise her left upper lid slightly.

During the months since her discharge, her pain has gradually disappeared completely, her proptosis has completely disappeared. The exophthalmometer reading is 18 mm. in both her eyes, with a base measurement of 104 mm. There no longer is any left upper lid droop. Meticorten therapy was discontinued on April 17, 1957. She can move her left eye well in all directions, except medially past the midline. This is true despite the fact the left medial rectus muscle was tucked and the left lateral rectus muscle was recessed on November 9, 1956. Because of no improvement a tenotomy of the right lateral rectus was performed on May 15, 1957. Neither of these muscle procedures improved the movement of the left eye past the midline nasally.

At the most recent visit (June 28, 1957) to the eye clinic, the Hertel exophthalmometer readings were still 18 mm. for both eyes with a vision of 20/20, O.U., with correction. No ptosis was present and she still was having no pain two months after steroid therapy had been discontinued. She could fuse straight ahead and to the left but still complained of seeing double when looking to the right. She was found to be orthophoric when looking to the left, to have 25 prism diopters of exophoria when looking straight ahead, and to have 50 prism diopters of exotropia when looking to the right.

Summary

The incidence of ocular involvement in sarcoidosis is fairly high. The incidence of orbital involvement in sarcoidosis is low. The diagnosis of sarcoidosis of the orbit should be suspected in patients with a painful unilateral proptosis and a palpable mass that can be felt through the eyelid. While steroid therapy appeared beneficial in the patient reported, the true value of this type of therapy in orbital sarcoidosis is still undetermined.

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PROCEDURE FOR REMOVING FOREIGN MATERIAL*

FROM THE POSTERIOR LAYERS OF THE CORNEA: WITH AN ILLUSTRATIVE CASE REPORT

C. TRUMAN DAVIS, M.D.
Temple, Texas

The removal of foreign material from the posterior layers of the cornea sometimes is difficult. If the material is friable, particles may fall into the anterior chamber during an attempt to remove the foreign body by forceps or by probing.

CASE REPORT

A 55-year-old farmer was seen in the clinic on March 19, 1956. About six weeks prior to admission, a thorn had penetrated his right eye. The thorn was removed by a general practitioner, and the eye seemed to respond until one week before the present examination. Redness, irritability, and sensitivity to light recurred; and in spite of conservative therapy, the symptoms persisted and became progressively worse.

Ocular examination revealed the left eye to be normal. Vision in the right eye was 20/30—2. There was marked injection of the globe, and a small penetrating corneal scar was visible at the 1-o'clock position about three mm. from the limbus. In the posterior layers of the cornea, a small mass of opaque foreign material appeared. From this mass, numerous fine filaments projected into the anterior chamber and actually moved slightly in the aqueous currents. There was a marked flare with numerous cells in the aqueous. It was believed that the foreign material was (1) vegetable fibers from the thorn, or (2) possibly a fungus mass growing from spores implanted in the corneal tissue by the thorn.

The inflammation of the anterior segment cleared rapidly with local treatment consisting of atropine, corticosteroids, and moist heat; however, any attempt to withdraw the steroid therapy resulted in a flareup of the iritis. To effect a cure, it was believed that removal of the foreign material from the posterior layers of the cornea would be necessary.

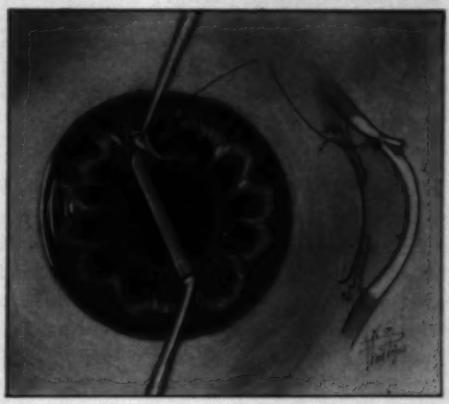

Fig. 1 (Davis). The corneal flap was hinged at the limbus.

Using topical anesthesia, the following operative procedure was performed:

A corneal flap, approximately three-mm. wide and four-mm. long, was raised with a Lunsgaard knife. This flap included approximately two thirds of the thickness of the cornea, and was hinged at the limbus (fig. 1). Black silk sutures (6-0) then were placed (fig. 2). Using a V. Mueller motorized trephine with a two-mm. blade, a button of posterior corneal tissue, including the foreign material, was removed (fig. 3). The corneal flap was sutured into place following removal of the corneal plug (fig. 4). The foreign material proved to be a fungus abscess with mycelia extending into the anterior chamber (fig. 5).

COMMENT

If the trephine incision is made perpendicularly to the surface of the cornea, the

* From the Department of Ophthalmology of the Scott and White Clinic.

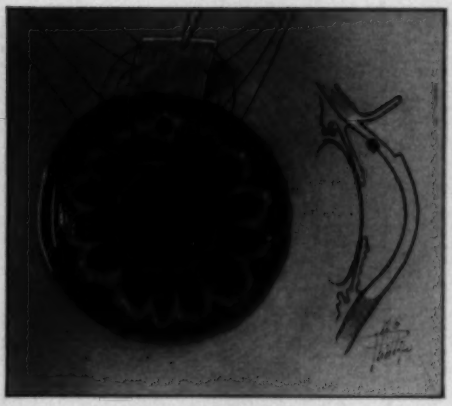

Fig. 2 (Davis). Black silk 6-0 sutures were placed.

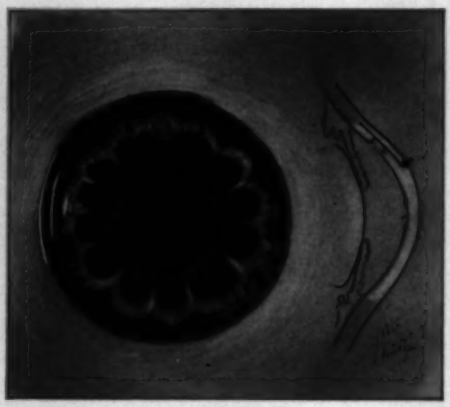

Fig. 4 (Davis). Following removal of the corneal plug, the corneal flap was sutured into place.

button will be cut smoothly around its entire periphery. The flow of aqueous humor through this opening then will force outward the corneal plug and any foreign material that is contained. Care must be taken not to exert pressure on the trephine during this procedure, as there is danger of injuring the anterior lens capsule when the anterior chamber collapses.

The patient's postoperative recovery was rapid and uneventful. Only very slight, transient edema of the stroma of the cornea occurred in the area of the incision. This cleared and, three months following treatment, a very faint opacity marked the area where the thorn had penetrated. The remainder of the cornea was completely clear. Eight months postoperatively, the inflammatory reaction had not recurred, and the patient had regained normal vision.

Scott and White Clinic.

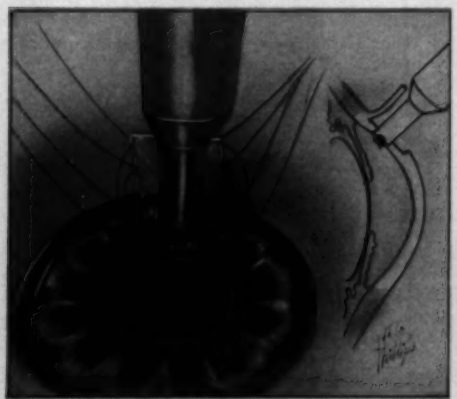

Fig. 3 (Davis). Using a motorized trephine a button of posterior corneal tissue was removed.

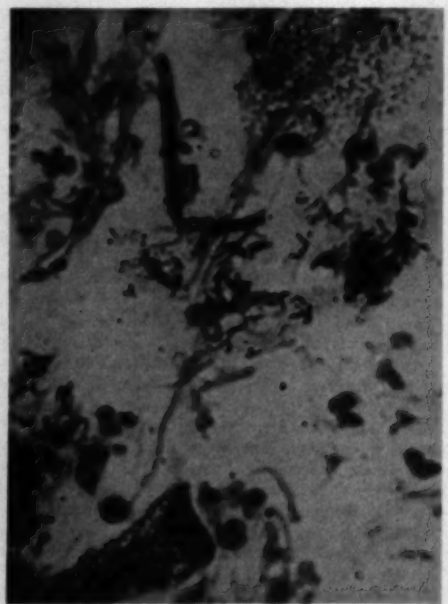

Fig. 5 (Davis). Microscopic view of fungus abscess.

MODERN ENGINEERING LOOKS AT KERATOPLASTY*

T. Elmer Moon and
L. Byerly Holt, M.D.
Winston-Salem, North Carolina

The collapse of the anterior chamber following the trephination of the cornea can create surgical complications. An orderly solution of certain of these surgical problems was sought through a group of instruments, so constructed that their application was mutually dependent upon one another, and so engineered that repetitively duplicate operations can be done in a uniform and standardized manner.

Over a period of 12 years many methods were tried, and finally it was found that the best solution for creating an "artificial platform" which would hold up the cornea while it was being trephined, was by the use of suction—and this feature has been incorporated (in its improved form) into what we may term a "vacuum eye cup." Each vacuum eye cup is contoured to fit the particular cornea which is being operated upon. The Marguerite Barr Moon Eye Research Foundation, Inc., is working on experiments which will determine the correct number of mm. Hg that are necessary not only to hold the cornea in position, but simultaneously so to distribute the suction area as to prevent injury to the cornea. From Figures 1 and 2 it can be seen that the vacuum eye cup has suction channels from the periphery along the concave surface to an aperture 6.5 mm. in width.

The flat surface around the upper part of the hollow cylinder is designed so that the shoulder of the trephine is limited in its downward motion, thereby controlling the depth of penetration through the layers of the cornea and into the anterior chamber.

Two good effects result from this design:

Fig. 1 (Moon and Holt). The vacuum cup.

(1) the trephine is automatically prohibited from deviating from that portion of the cornea which the surgeon had selected by confining the area within the 6.5-mm. aperture incorporated in the vacuum eye cup; (2) the depth of penetration into the anterior chamber is automatically limited. Actually there is from 1.5 to 2.5 millimeters clearance between the greatest depth of trephine penetration and the iris.

Figure 3 shows a complementary device which we call the "arbor." There are a number of small holes as a continuation of the contoured surface and this specific portion serves to hold the donor's disc after it has been trephined. A pin, which projects from

Fig. 2 (Moon and Holt). Another view of the vacuum cup.

* From the Marguerite Barr Moon Eye Research Foundation, Inc. Aided by a grant from the National Institutes of Health, Bethesda, Maryland, under Contract Number B-1243.

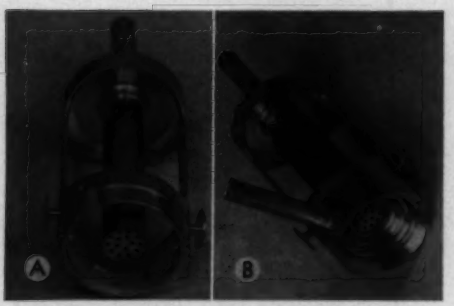

Fig. 3 (Moon and Holt). A complementary device called the "arbor."

the central tubing, is provided upon which to hang the trephine. This trephine has a bayonet joint which permits the surgeon to keep the instrument out of his way until such time as he wishes to use it. When he is through with this phase of the operation he can hang up the trephine while he removes the "arbor" from its nesting place in the vacuum eye cup.

In a similar manner a uniform and standardized disc may be removed from the recipient's eye. With one vacuum eye cup still holding up the cornea of the recipient's eye the disc removed from the donor's eye is transferred to its precise aperture in the recipient's eye. The vacuum is then slowly reduced in the arbor (which holds the donor's disc) and a final deft squeeze by the surgeon on the vacuum tube releases it. By means of the precision apertures, the donor disc is accurately aligned with the corneal tissue in the recipient's eye and any need for trimming or cutting with scissors is completely eliminated.

209 Reynolds Building (3).

Ophthalmic Miniature

I could not procure a sufficient quantity of these (human eyes), fresh enough to multiply my experiments upon them. However, by the assistance of Mr. Carpue, surgeon to his Majesty's Forces, I fully convinced myself, that the humours of the human eye, chemically considered, did not contain anything different from the respective humours of the (animal) eyes I had examined. The aqueous and vitreous humours contained water, albumen, gelatine, and muriate of soda; and the crystalline humour contained only water, albumen and gelatine. The specific gravity of the aqueous and vitreous humours, I found to be 10053; while that of the crystalline was 10790.

R. Chenevix,
"Observations on the chemical nature of the humours of the eye,"
Read before the Royal Society on November 5, 1802.

SOCIETY PROCEEDINGS

Edited by DONALD J. LYLE, M.D.

COLLEGE OF PHYSICIANS OF PHILADELPHIA

SECTION ON OPHTHALMOLOGY

March 21, 1957

DR. ISAAC S. TASSMAN, *Chairman*

TEMPORAL ARTERITIS

DR. PAUL DAVIDSON (by invitation), DR. NATHAN S. SCHLEZINGER, AND DR. CARROLL R. MULLEN: Since temporal arteritis is a disease that responds to appropriate treatment, early diagnosis is necessary in order to prevent the onset and progression of its most serious sequel, blindness. This is exemplified by a 67-year-old woman who rather suddenly lost the vision of her right eye seven weeks following the onset of incapacitating headaches. The significant findings initially were: a febrile reaction reaching a level of 102°F.; tenderness over the temporal and occipital arteries; blindness of right eye with pallor of the right optic disc, and attenuated retinal arterioles. The condition failed to improve with antibiotic therapy and low dosage steroid therapy. A progressive course included near loss of vision of the left eye and episodic mental confusion.

The establishment of the diagnosis of temporal arteritis and the administration of cortisone in a dose of 300 mg. daily resulted in prompt and dramatic remission of all symptoms except impaired vision. A biopsy of the temporal artery showed complete occlusion with characteristic alterations in the vessel wall including many giant cells in the media.

Temporal arteritis is an acute form of "giant cell arteritis" of unknown etiology. There is no sex predilection. The prodromal symptoms may resemble rheumatoid arthritis. Although any artery in the body may be affected, there is most often involvement of the temporal and retinal arteries. Ocular involvement occurs in about 40 percent of cases with equal incidence in this group of unilateral and bilateral blindness. The visual loss is almost completely irreversible. The most effective treatment thus far has been cortisone therapy in large doses. Apparently this is able to prevent the onset and progression of blindness but will not restore vision that is lost.

Discussion. DR. NATHAN S. SCHLEZINGER: I think the presentation has been comprehensive enough probably to make much discussion unnecessary. I think the main purpose is served by bringing to the attention of those here the problem of temporal arteritis in terms of early diagnosis, and proper treatment in order to avoid the unfortunate, tragic advent of blindness. Now in this particular patient, I think we would want to recognize how, at first glance, an infectious disease would be seriously considered.

In retrospect in the study of this patient, one would wonder what kind of an infectious disease would be present with a normal blood count. A normal blood count and a rapid sedimentation rate are commonly observed in a collagen disease. This was the type of patient who is sick enough when first observed in the hospital to make it difficult to obtain accurate details with regard to her symptoms. These were, in general, obtained later. She had mental symptoms which not infrequently are observed in serious cases of temporal arteritis. The therapy, which initially was directed toward an infectious disease, did not, in my opinion, include adequate cortisone.

Of course, when the diagnosis was established, immediate and dramatic results were obtained. Within a period of 12 hours this patient became asymptomatic in terms of her profound general manifestations. The head-

aches were almost gone. The vision was somewhat improved in the eye most recently involved. There was continued improvement, although the patient required a rather large amount of cortisone therapy. In looking back, it would seem that the eyes were affected after corticoid therapy, administered over a period of a year or longer, was stopped when this patient developed an ear infection. It is likely that a continuation of cortisone therapy might have prevented the exacerbation which involved the retinal vessels and affected the vision. One should always bear in mind that individuals who are under prolonged corticoid therapy may be potential subjects for temporal arteritis. Whenever such therapy is terminated, one should be alert for the development of a temporal arteritis syndrome. I believe I have seen in consultation some "burned out" cases of unrecognized temporal arteritis. Ophthalmologists should review any cases that might show the clinical picture presented here this evening. Early diagnosis is essential because, when blindness has developed, it is too late to expect results from corticoid therapy.

DR. I. S. TASSMAN: It is difficult to recognize temporal arteritis and important to make an early diagnosis. After the eyes are involved, the visual impairment is usually permanent, as it was in this case. This particular patient presented symptoms which gave little reason to think of temporal arteritis until rather late in the course of the disease. I would just like to ask whether the patient is still under treatment?

DR. PAUL DAVIDSON: Yes, the patient is still receiving 200 mg. cortisone a day, and her doctor has been unable to reduce the dosage because of exacerbations.

William E. Krewson, 3rd,
Clerk.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

436th Meeting, March 20, 1957

DR. EDWARD A. CRAMTON, *presiding*

CASE PRESENTATIONS

Case 1—Tumor of the eye. DR. ALFRED W. SCOTT: I think the history of this woman is somewhat misleading. On Friday she had a headache over the right eye. On Saturday morning when she awakened, she noticed a blur in the right eye. This blur has fluctuated a little since that time. It was also noted on Saturday morning that the conjunctiva was definitely red, not markedly so, however. This has continued. When I examined her in the office on Tuesday, she had vision of 20/20 with pinhole. The media were clear. The conjunctiva of the right eye was about one- or two-plus injected but not on the left. The anterior chamber was clear, with no cells or flares. The pupils were equal in both eyes. There was an absolute field defect in the lower outer quadrant. Corresponding to this, there was a lesion in the upper nasal quadrant. This lesion had two or three small hemorrhages on its surface. This is why I bring her in for diagnosis. *Consensus:* Malignant melanoma.

Case 2—Melanotic sarcoma of the iris. DR. GARRETT L. SULLIVAN presented a 41-year-old man. Twenty years ago a tiny spot was noted on the iris of the right eye. Fifteen years ago, when the man was inducted into the Army, the spot was noted by the Army doctors. In recent years this spot has grown larger. The man has noticed no interference with vision.

On examination, he now shows a mushroom-shaped, brownish-black lesion in the iris at the 6-o'clock position, coming almost to the pupillary border. The pupil is drawn very slightly at the 6-o'clock position and there is definite limited mobility of the pupil at this point. Fundus examination shows posterior cortical lens opacities, superiorly.

These do not involve the central portion of the lens. A limited view of the fundus shows normal disc and vessels. Vision in the right eye is 20/30, correctible with pinhole to 20/20. Uncorrected vision in the left eye is 20/15.

Under the corneal microscope the lesion is noticeably elevated and has rather a bumpy surface. There are no vessels to be observed in the vicinity of the lesion. Under gonioscopy, the elevated area appears to slope off into the angle and to extend out to about the 3-o'clock position on one side and around to the 10-o'clock position on the opposite side. There was noticeable pigmentation in the angle, consisting of varied sized dark, black spots; most of these were on the extreme face of the cornea. There were many little fine iris frills going toward the cornea.

Tension was 17 mm. Hg (Schiøtz). The other eye is completely normal.

I feel the diagnosis is melanotic sarcoma and that the eye should be enucleated. I would welcome any opinions.

Discussion. DR. FREDERICK H. VERHOEFF: I used to think, when I saw a tumor like this, that the only thing to do was to take the eye out. But so many of these cases have gone for such a long time without anything happening that I have begun to wonder if I was right about it. Wouldn't it be a good idea in this particular case just to watch for awhile? Measure the lesion. You are not sure that it has grown, are you? Nobody has measured it, have they? I should think it would be worth while to do that.

I think the patient ought to be told that the only absolutely safe thing to do is to take his eye out. But then I would tell him if it were my eye I wouldn't have it out right away. I say this because of the records of these cases. This is really a small tumor. On the skin, as a mole somewhere, it would be a very small mole. A lot of them have gone on a long time. In measuring them, do not worry too much about great accuracy. Take a pair of calipers and approximate the thing; this should tell if it is growing; or take a series of pictures over a period of time and compare them.

DR. EDWIN B. DUNPHY: This might be a good case in which to try a P^{32} test. The lesion seems to be fairly accessible.

CATARACT EXTRACTION AND TONOGRAPHY AND GONIOSCOPY

DR. PEI-FFI LEE: Tonographic and gonioscopic studies before and after cataract extraction offer much valuable information in handling postoperative cases with and without complications.

Postoperatively the facility of outflow may be temporarily decreased, increased, or unchanged. With temporarily decreased outflow, there often is definite elevated tension. Most of these eyes do not require any antiglaucomatous therapy but should be watched closely and carefully in order to prevent unnecessary damage.

In our series the facility of outflow of patients with pseudoexfoliation, glaucoma capsulare, and open-angle glaucoma was essentially unchanged before and after the cataract extraction. On the other hand, the cataract extraction did not help the open-angle glaucoma symptoms so far as intraocular pressure and tonographic findings were concerned.

The deposition of pigment at the inferior angle, transparent vessels on the surface of the peripheral iris, and the discoloration of the posterior meshwork are purely senile phenomena. However, in a patient with the later stages of diabetes mellitus combined with advanced cataract, preoperative gonioscopic examination is necessary in order to detect abnormal vessels and to warn of the danger of hemorrhage.

About 95 percent or more of the inner scars were located anteriorly in the cornea and, postoperatively, did not interfere with maintenance of a normal intraocular pressure and corrected vision. Separation of Descemet's membrane often occurred at the area where the surgical wound was extended with scissors.

In handling patients with flat anterior chambers postoperatively, gonioscopic examination should be done after the anterior chamber is reformed and the indicated therapy is being given.

Corneal dystrophies and degenerations

Dr. David D. Donaldson: I would like to say something about the definitions of dystrophies, degenerations, and keratopathies. These terms are used for all the noninflammatory conditions of the cornea. Generally speaking, we think of a dystrophy as being a condition in which there is an hereditary factor; there is no other known cause. Degenerations are associated with old age or with eyes in which the vitality of the tissues is decreased. It is often difficult to differentiate between dystrophies and degenerations. I might also say that a number of dystrophies are really degenerations. Keratopathies are those corneal conditions that have a definite cause or are secondary to some systemic condition.

The classification I have worked out for dystrophies and degenerations is a modification of the system proposed in 1950 by Franceschetti. I have added to it and changed it a bit to fit the ideas I have.

The first general group is termed "Parenchymatous." Under this heading are first the classical forms which include granular dystrophy (Groenouw's type I); lattice dystrophy; and macular dystrophy (Groenouw's type II). The second "Parenchymatous" group includes the congenital forms of posterior embryotoxon of Axenfeld and sclerocornea. In the last group are the diverse forms of crystalline dystrophy; lipoid dystrophy; furrow dystrophy; arcus senilis (gerontoxon); arcus juvenilis (anterior embryotoxon); keratoconus; and cornea farinata.

The second general group is placed under the heading of "Limiting zones." In this grouping, the anterior group includes Salzmann's nodular "dystrophy"; band-shaped dystrophy; Vogt's limbus girdle; Coats' corneal ring; crocodile shagreen (anterior); primary essential corneal edema; juvenile epithelial degeneration; and recurrent hereditary erosion. The posterior group includes cornea guttata ("epithelial" degeneration of Fuchs); crocodile shagreen (posterior); and posterior polymorphous degeneration.

The last general group is termed "Corneal degenerations in general affections." Under this heading are the metabolic disturbances of Hurler's disease; cystinosis; Hand-Schüller-Christian disease; and ochronosis. Under the heading of "Cutaneous conditions" are ichthyosis; Rothmund's syndrome; and keratoma palmare et plantare hereditarium.

Charles Snyder,
Recorder.

YALE UNIVERSITY CLINICAL CONFERENCES

November 9 and 16, 1956

Dr. Rocko M. Fasanella, *presiding*

Psychologic considerations in surgery

Dr. Edith Jackson and Irving Janis, Ph.D., presented a very interesting discussion of the psychologic aspects in patients undergoing surgery. At this time one is confronted with a problem in emotional inoculation, that is, preparing a person for a situation of stress. A purely intellectual approach may not be very successful. Three aspects were discussed:

1. How do people with different levels of preoperative anxiety react in the postoperative situation?

2. Does preoperative information affect postoperative behavior?

3. How is the stress before surgery correlated with other stress situations, as in combat, and so forth?

In connection with the first point, were three general types of reactions to exposure to threat of a dangerous situation: (a) fear,

(b) resentment and aggression, (c) emotional depression. The problem was studied in two ways: (1) intensive case studies and (2), a large scale survey of 1,000 Yale students.

In the case studies, the patients were classified according to their level of preoperative fear and anxiety: the low fear group, the moderate, and the high fear group. These were all classified both by their own statements and by their actions preoperatively. The cases with a high level of preoperative fear showed a high level of fear during and after surgery and a similar situation held for the medium fear cases. The cases with low fear levels preoperatively showed moderate levels during and after surgery. Regarding the resentment factor, however, the cases with low fear preoperatively showed a higher resentment postoperatively than even the middle or high fear cases did. In concluding the case studies, it was felt that the high-fear cases are neurotically predisposed patients. The other two groups are not.

Cases with low preoperative fear level were very badly adjusted after operation. They often felt they were cheated, that they didn't deserve what happened to them, and so forth. The Yale questionnaire studies in general supported these conclusions. There was often a fear of being alone or being abandoned by loved ones. Patients often felt very grateful postoperatively and that the operation was very successful, but any renewed threat often served to reawaken a high level of fear. In general, those patients with a moderate level of preoperative fear reacted best postoperatively.

In regard to the effect of preoperative information, it was believed that, in many cases, low fear levels were due to low information levels, especially with the popular types of operation, such as appendectomy, in which there was absolutely no knowledge of the experience to be encountered—postoperative discomfort, and so forth.

In conclusion, it was noted that the capacity of a patient to resist stressful situations depends on how well, subjectively, the unpleasant experiences have been worked before hand and whether a reaction has developed before the actual situation arises. Discomfort postoperatively is less likely to arouse resentment if the patient is prepared; if unprepared, there is a much greater tendency to a sensitizing effect.

Discussion. DR. JACKSON: There are many applications of Dr. Janis's studies in the field of pediatrics. Many adult reactions are childlike. Many parents deny to the child that anything unpleasant is going to happen and this is a very poor way of preparation.

DR. RUBINSTEIN: One should be a little suspicious of a patient who denies all anxiety and fear preoperatively. This patient may really be one with a high level of preoperative fear.

DR. FREEMAN: The first night spent in the hospital preoperatively may be very traumatic and that is a good time to prepare the patient for operation.

DR. SIMEL: How do adults undergoing local anesthesia differ in their reactions from those undergoing general anesthesia?

DR. JANIS: With patients under local anesthesia, there is a severe period of stress during the operation itself in addition to the pre- and postoperative periods. It frequently is an irrational fear.

DR. FASANELLA: What is the feeling toward having parents with their children before and after operation.

DR. JACKSON: I am sympathetic to the trend of having parents in the room with young children. Fear is mostly associated with separation from parents. In the child of four years or younger, this is especially important. It is also helpful to reduce, as much as possible, the preoperative period in the hospital. After the age of five years, explanations can be better accepted. For a young child, certain explanations of what will happen when he awakens are important. Recovery room time may mean a period of loneliness which needs preoperative explanation.

DR. JANIS: The function of information to the patient in general is to enable him to cope with postoperative situations and experiences, to try to get him to work through these situations and develop self-reassurance prior to the actual experience. In this connection, it is also possible to give him reassurance of which he is not aware.

DR. GLASS: There is a certain type of patient with whom we all have come in contact and for whom I do not believe it is desirable to go into specific details in connection with preoperative preparation. Anxiety level is so high that any one statement may awaken two or three other questions in the patient's mind. After 15 or 20 minutes of "reassurances," the patient may be more disturbed than before the explanations were started. I believe, in this type of patient, general reassurances only are advisable and one should shy away from very specific statements about the nature of the operation and postoperative discomfort.

DR. JANIS: That is quite true and these patients of the very high preoperative fear level may even tend to have psychiatric problems and irrational fears.

REOPERATION AND COMPLICATIONS IN GLAUCOMA

DR. EDMUND B. SPAETH (Philadelphia): Operations for glaucoma have a rather definite incidence of unsuccessful results. Consideration of reoperation is, therefore, of serious importance. Reoperation, itself, has such a high percentage of failures that it is quite necessary to evaluate every case very carefully.

Four different basic methods of lowering tension surgically were discussed: Iridectomy, intraocular drainage operations, extraocular drainage operations, and operations to decrease aqueous formation.

The problems of reoperation upon eyes unsuccessfully operated on were then considered. These problems vary according to how soon after the original operation it is decided that a second operation is necessary. For instance, an immediate failure of a cyclodialysis will contraindicate another cyclodialysis at that time because of the ciliary irritation. A late failure of cyclodialysis could very well be followed by another cyclodialysis in a different region of the eye.

Dr. Spaeth then spoke about true complications of glaucoma surgery, such as hemorrhage, lens-capsule damage, rupture of the zonule, delayed reformation of the anterior chamber, posterior synechias, and cataract. The methods of avoiding these problems and handling them when they did occur were discussed in detail.

Discussion. DR. LOVEKIN: I would like to emphasize the discovery of early cases so that favorable surgery is possible where indicated, rather than having to work on unfavorable cases. Postoperatively, I usually do not advise the use of pilocarpines but prefer mydriatics and cycloplegics.

DR. SPAETH: The question of early diagnosis brings up the controversy of medical versus surgical treatment. There are no universal rules here. I feel that not all glaucomas are surgical diseases.

DR. VAN HEUVEN: (1) What surgery would you advise in cases with anterior synechias following perforating wounds of the cornea? (2) How much cyclodiathermy do you do at the first operation? (3) What method of disinfection do you use in preoperative preparation? In fistulizing operations, the sooner they are done, the better the prognosis if any field loss develops. However, what is your feeling if the fields are very small?

DR. SPAETH: An anterior synechia after corneal perforation is responsible for keratitis and progressive corneal opacity. Knife needle rupture and air injection are usually necessary. In cyclodiathermy, I make the application 6.5 mm. behind the limbus for a duration of eight seconds each, applying a total of 12 applications at the first operation. I may follow it with the paracentesis and an

air injection to be a buffer against the temporary rise in pressure that occurs postoperatively.

My present method of preoperative preparation is a soap and water scrub and a drop of silver nitrate followed by irrigation. In connection with the problem of surgery in a patient with contracted fields, the surgery affects the pressure relations in the capillaries of the remaining good retina and I, therefore, try to prevent an abrupt decompression of the anterior chamber when operating, but I would go ahead with the operation when otherwise indicated.

DR. GLASS: Under what circumstances would you use an iridencleisis in a case of acute glaucoma? Also, have you seen any cases of blood-staining of the cornea develop when the tension was high but the hyphema was not a complete one?

DR. SPAETH: Iridencleisis in acute glaucoma should probably be done when there is a history of glaucoma and also when the acute glaucoma has been present for three to six days prior to surgery. However, it is not routinely necessary in all cases. In connection with blood-staining of the cornea, I haven't seen it develop unless the anterior chamber was pretty full of blood.

DR. VERNON: Do you favor the use of peripheral iridectomy?

DR. SPAETH: Peripheral iridectomy is a sound procedure. I think it is important in an early case when it is felt that the development of glaucoma is definite but the aqueous outflow facilities are in the indeterminate range.

William I. Glass,
Recording Secretary.

OPHTHALMIC MINIATURE

The advantages of the corneal flap extraction may be much enhanced, and its dangers materially lessened, in my judgement, by the use of a suture to retain in apposition the edges of the wound. Securing a more immediate union, we not only avoid ulceration of the border of the flap, and prolapsus iridis with its attendant evils, but the prompt restoration of the fullness of the globe, and of the normal relations of its several parts, lessen the chances of irritation, from pressure of any cortical fragments or remnants of capsule upon the delicate contiguous structures, and the occurrence of irido-cyclitis. This suture, a single strand only of the finest glover's silk, passed through the edge of the wound by means of a very minute, short needle, held by forceps, can be tolerated, without detriment, even in the cornea.

"Cataract extraction operations,"
Henry W. Williams,
Archives of Ophthalmology and Otology, 1:102, 1869.

PROGRAM

of the

ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY

27th Annual Meeting

Terrace Room Fairmont Hotel

San Francisco

June 23-27, 1958

Monday morning, June 23, 1958

8:30 An electromyographic study of asymmetric convergence. Edward Tamler, Arthur Jampolsky, and Elwyn Marg, San Francisco.
Discussion to be opened by F. C. Blodi.

9:00 The electromyographic pattern of saccadic eye movements. James E. Miller, St. Louis, Missouri.
Discussion to be opened by F. C. Blodi.

9:20 Heterologous immunologic studies with lens. Patricia FitzGerald and Seymour P. Halbert, New York.
Discussion to be opened by Phillips Thygeson.

9:40 Radioelectrophoretic patterns of aqueous and plasma after intravenous injection of I^{131}-labelled insulin into normal and diabetic rabbits. Kenneth M. Giles and John E. Harris, Portland, Oregon.
Discussion to be opened by John W. Patterson.

10:00 11-Cis vitamin A in the prevention of retinal rod degeneration: an animal study. Albert Chatzinoff, William Oroshnik, Nathan Millman and Fred Rosen, New Hyde Park, Raretan, and Buffalo.
Discussion to be opened by Albert M. Potts.

10:20 Intermission

Group discussion of the following papers, to be moderated by Albert M. Potts.

10:40 Electrical activity of cells in the eye of limulus. M. G. Fuortes, Bethesda.

11:00 Conduction velocities in rabbit's optic nerve and their relationship to retinal spikes. Ragner Granit and Elwin Marg, Stockholm, Sweden, and San Francisco.

11:20 The electrical activity of the eye during accommodation. Jerry H. Jacobson, Hunter H. Romaine, G. Peter Halberg and George Stephens, New York.

11:40 Changes in spontaneous and evoked potentials on the electroretinogram induced by drugs: Strychnine, Atropine, LSD-25, Mescaline. Julia T. Apter, Chicago.

Monday afternoon, June 23, 1958

2:00 Mechanism of corneal destruction by Pseudomonas proteases. Earl Fisher, Jr., and James H. Allen, New Orleans.
Discussion to be opened by Daniel Vaughn.

2:20 Toxoplasmosis: The nature of virulence. Herbert E. Kaufman, Jack S. Remington, and Leon Jacobs, Bethesda.
Discussion to be opened by Samuel Kimura.

2:40 Studies on chronic toxoplasmosis: The relation of infective dose to residual infection and to the possibility of congenital transmission. Jack S. Remington, Leon Jacobs, and Herbert E. Kaufman, Bethesda.
Discussion to be opened by Michael J. Hogan.

3:00 In vitro observations on the behavior of conjunctival and corneal cells in relation to electrolytes. John Y. Harper, Jr., Galveston.
Discussion to be opened by Marguerite A. Constant.

3:20 Physiologic studies of the developing chick cornea. Alfred J. Coulombre, New Haven.
Discussion to be opened by George K. Smelser.

3:40 Intermission

4:00 Antigen-antibody reaction in the rabbit cornea. C. E. van Arnam, John Pratt-Johnson, Alfred E. Maumenee and Frederick G. Germuth, Jr., Baltimore.
Discussion to be opened by Phillips Thygeson.

4:20 The effects of various constituents of a synthetic medium on cell division in the epithelium of cultured lenses. Carl Wachtl and V. Everett Kinsey, Detroit.
Discussion to be opened by Hugh L. Ormsby.

4:40 A tissue culture technique for growing corneal epithelium, stroma and endothelium separately. Frederick W. Stocker, A. Ewing, R. Georgiade and N. Georgiade, Durham, North Carolina.
Discussion to be opened by Carl Wachtl.

Tuesday morning, June 24, 1958

8:30 Electron microscopy of the ciliary epithelium and zonula of the rabbit. George D. Pappas, and George K. Smelser, New York.
Discussion to be opened by Parker Heath.

9:00 The application of ultrasonic locating techniques to ophthalmology. Gilbert Baum, Port Chester, New York.
Discussion to be opened by Howard M. Yanof.

9:20 Protein dynamics in the eye studied with labelled proteins. David M. Maurice, San Francisco.
Discussion to be opened by Harry Green.

9:40 Changes in proteins and protein synthesis in lenses of rats during the development of tryptophan and radiation cataract. Zacharias Dische, Joy F. Elliott and George R. Merriam.
Discussion to be opened by Jin Kinoshito.
10:00 Intermission
10:20 The influence of methylene blue and other dyes on the cation and water balance of the lens. John E. Harris, Louise Gruber, and Gertrude Hoskinson, Portland, Oregon.
Diseussion to be opened by Bernard Becker.
10:40 Additional observations on the lenticular potential. Nick Sperlakis and Albert M. Potts, Cleveland.
Discussion to be opened by D. Venkat Reddy.
11:00 In vitro lens studies: III. Lens potential. Marguerite A. Constant, St. Louis.
Discussion to be opened by Jin Kinoshito.
11:20 Corneal transport of tritiated water. Albert M. Potts and Beatrice Cohen, Cleveland.
Discussion to be opened by Frank Newell.
11:40 Ocular changes induced by polysaccharides: II. Detection of hyaluronic acid sulfate after injection into ocular tissues. Ellen Talman, John E. Harris, and Louise Gruber, Portland, Oregon.
Discussion to be opend by Zacharias Dische.

Wednesday morning, June 25, 1958

8:30 Elevation of choroid by insertion of polyvinyl sponge: An experimental study. Martin S. Kazdan and John W. Henderson, Rochester, Minnesota.
Discussion to be opened by William G. Everett.
9:00 Observations on accommodative convergence especially with regard to nonlinear relationships. T. G. Martens and Kenneth N. Ogle, Rochester, Minnesota.
Discussion to be opened by Gerhard A. Brecher.
9:20 The effect of topical application of certain so-called autonomic drugs on human visual flicker discrimination. Mathew Alpern and Robert S. Jampel, Ann Arbor.
Discussion to be opened by Paul W. Miles.
9:40 Studies on the anatomy and pathology of the peripheral cornea. John A. Pratt-Johnson, San Francisco.
Discussion to be opened by A. Ray Irvine, Jr.
10:00 Intermission
10:20 Business meeting
10:40 Distribution of acid mucopolysaccharides in the developing mouse eye: A study of pigmented and albino strains including the rodless C_3H mouse. Lorenz E. Zimmerman and Ann B. Eastham, Washington, D.C.
Discussion to be opened by George Smelser.
11:00 The Friedenwald Memorial Lecture. Bernard Becker.

Thursday morning, June 26, 1958

8:30 Lacrimal protein patterns. Olive F. Erickson, San Francisco.
Discussion to be opened by Peter C. Kronfeld.
9:00 Incorporation of radioactive sulfate in the guinea pig eye: Influence of hormones and avitaminosis: An autoradiographic investigation. Godfred Larsen, New York.
Discussion to be opened by Frank Newell.
9:20 Elaboration of the bicarbonate ion in intraocular fluids: VI. Kinetic studies with C^{14}-labelled $NaHCO_3$. Harry Green, John L. Sawyer and Samuel I. Askovitz, Philadelphia.
Discussion to be opened by V. Everett Kinsey.
9:40 Outflow patterns of the cat eye. Frank J. Macri, Bethesda.
Discussion to be opened by Robert Moses.
10:00 Intermission
10:20 The effects of beta radiation on the aqueous secretions in rabbits. Frank S. Schiff, Alhambra.
Discussion to be opened by Peter Kronfeld.
10:40 Pressure-volume relationship in the intact human eye. Robert A. Moses and Ahti Tarkkanen, St. Louis.
Discussion to be opened by Earl McBain.
11:00 Proctor Medal Recipient
Alternate Paper: The hydrophilic property of the orbital connective tissue of normal and exophthalmic animals. George K. Smelser and Victoria Ozanics, New York.

PROGRAM

of the

SECTION ON OPHTHALMOLOGY

AMERICAN MEDICAL ASSOCIATION

Fairmont Hotel San Francisco

June 24-26, 1958

Tuesday afternoon, June 24, 1958

Chairman's address. Dohrmann K. Pischel, San Francisco.

Address of invited foreign guest. Indications for clinical electroretinography. Prof. Gosta Karpe, Stockholm, Sweden.

Conjunctival flaps in the treatment of corneal disease: With special reference to new techniques of application. Trygve Gundersen, Boston.

Discussion to be opened by Phillips Thygeson.

The treatment of retinoblastoma by X-ray and triethylene melamine. Algernon B. Reese, George A. Hyman, Norah duV. Tapley and Arnold W. Forrest.

Discussion to be opened by Michael J. Hogan.

Galactosemia with associated cataracts in children. Warren A. Wilson, Los Angeles.

Discussion to be opened by Harold F. Falls.

The neurosurgeon's role in acute visual failure. Alfred Uihlein and C. Wilbur Rucker, Rochester, Minnesota.

Discussion to be opened by William E. Krewson, III.

Ocular signs and prognosis in subdural and subarachnoid bleeding in young children. Robert W. Hollenhorst, and Harold A. Stein, Rochester, Minnesota.

Discussion to be opened by Donald J. Lyle.

Wednesday afternoon, June 25, 1958

Executive session

The prognosis of melanomas of the iris following excision by iridectomy: A study of 108 cases in the registry of ophthalmic pathology. Benjamin Rones and Lorenz Zimmerman, Washington, D.C.

Discussion to be opened by Georgiana Dvorak-Theobald.

Diseases of the macula: Basic histopathologic processes in retina, pigment epithelium and choroid, which modify their clinical appearance. Bertha A. Klien, Chicago.

Discussion to be opened by William C. Frayer.

Clinical and laboratory experiences with virac-A bactericidal, fungicidal and viricidal agent. John E. Harris, Peter P. Rowell and Olive Beaudreau, Portland, Oregon.

Discussion to be opened by Henry F. Allen.

The effect of tonography and other pressures on the intraocular blood volume. Jerome W. Bettman, Victor Fellows and Peter Chao, San Francisco.

Discussion to be opened by Bernard Becker.

Thursday afternoon, June 26, 1958

PANEL ON RETINAL DETACHMENT

A scleral imbrication technique. Albert N. Lemoine, Jr., James T. Robison, Jr., and Larry T. Calkins, Kansas City.

An evaluation of ophthalmoscopy after retinal detachment surgery. Ariah Schwartz, San Mateo, California.

Retinal detachment surgery. James S. Shipman, Camden, New Jersey.

The old versus the new. John W. Henderson, Rochester, Minnesota.

The importance and employment of diathermy in today's retinal detachment surgery. Graham Clark, New York.

Further experience with vitreous implants in old retinal detachments. Donald M. Shafer, New York.

Diathermy or scleral resection. Joseph A. C. Wadsworth, New York.

The application of ultrasonic locating techniques to ophthalmology: Part II. The ultrasonic slit lamp. Gilbert Baum, Bronx, New York.

Discussion to be opened by: S. Rodman Irvine, Beverly Hills; Peter C. Kronfeld, Chicago; P. Robb McDonald, Philadelphia, and A. D. Ruedemann, St., Detroit.

AMERICAN JOURNAL OF OPHTHALMOLOGY

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THE HADLEY SCHOOL FOR THE BLIND

It is unfortunate but true that the ophthalmologist is usually ill prepared to advise the blinded patient and guide him along those paths which may best enable him to lead a happy and useful life. Psychologically this can be well understood since, as ophthalmologists, we are concerned with measures calculated to prevent the onset of blindness. When loss of sight occurs in our patients in spite of all the medical and surgical measures employed, there is a natural feeling that we have reached the end of our professional usefulness. We do, however, have a moral obligation as physicians first to assist the patient in the mental adjustment to his new state and then to advise him as to

the various agencies available to help him in the process of rehabilitation as a blind individual in a seeing world.

Since Valentin Haüy opened the first school for the blind in Paris in 1784, there have been tremendous advances in the techniques employed in education of the blind. Louis Braille, a blind teacher of the blind, may be regarded as the patron saint of the method of reading raised dots through the fingers, although his method was not the only nor indeed the first method proposed but, after a pitched "battle of the types," it became almost universally adopted. The invention and development of the phonograph, especially of the long-playing record, has been a boon to the blind, especially to that large percentage which is not able to master the intricacies of braille. Pioneered by the American Foundation for the Blind and introduced in 1934 as the "Talking Books" some 4,000 of these records have been available, with reproducers, for certified blind individuals and distributed, postage free, through the Library of Congress and its agents. Also available from this same source are some 25,000 books of fiction, biography, and popular nonfiction printed in braille. No textbooks are included in this collection. The development of records playing at even lower speeds than 33 rpm and the wider use of tape recording hold promise of ever-increasing horizons in literature for the blind.

It is estimated that in the United States alone there are some 200 organizations concerned with rehabilitation and education of the nation's estimated 330,000 blind individuals. This excessive number of Federal, State, and private agencies attests to a wide interest in this field on the part of the public but also suggests some needless duplications which doubtless will be solved eventually by mergers, amalgamations, and discontinuances. It is obviously impossible for the ophthalmologist to be familiar with all of these agencies but he can and should acquaint himself with those most effectively serving his particular community.

A unique educational institution offering free correspondence courses for the blind is the Hadley School which, in January, opened its new $200,000 building in Winnetka, Illinois. When William A. Hadley, a Chicago schoolteacher, became blind in 1916 he taught himself braille and then went on to interest others in a proposal to convert textbooks into braille and to make these available by mail to students throughout the world. With encouragement from his many friends and especially urged on by the late Dr. E. V. L. Brown, the Hadley School for the Blind was incorporated in 1922 as a not-for-profit institution. From the first course in beginning braille offered to two students in 1920 the school has expanded to the point where approximately 75 correspondence courses are offered, including four college courses for which credit is given by the University of Chicago. As of December, 1957, there was a total of 1,103 students registered from 51 states and territories of the United States and 174 students from 41 foreign countries.

Support by the local Community Chest, United Funds, Lions Clubs, and donations by interested citizens have made possible the expanding program of free correspondence courses. About one third of one percent of the school's income is derived from donations from grateful students. The school's beautiful new modern building will house about half of its staff of 20, a three-level library for its large collection of braille textbooks, and a modern recording studio which will be utilized for preparing records and tapes for courses and for individual correction of lessons returned to the students. A wide variety of courses is offered, all free and all by mail. There are included such subjects as braille (in which about 500 are registered), English grammar, spelling, composition, history, civics, algebra and arithmetic, six classical and modern languages,

sciences, salesmanship, music appreciation, and poultry raising.

We offer congratulations to the Hadley School on its new home and best wishes for continued success in its field as the only free correspondence school for the blind of the world.

William A. Mann.

CORRESPONDENCE

Facilities for the visually handicapped

Editor,

American Journal of Ophthalmology:

Recently it was brought to my attention by one of our local social agencies that few practicing ophthalmologists are conversant with the proper handling of the person with a permanent visual handicap. The agency further pointed out that few medical schools have this important phase of patient management in their curricula. Although our educational programs undoubtedly are doing a better job of training men in the science of diseases of the eye, they evidently have been remiss in teaching the art of handling the patient whom medical science cannot help.

What usually happens, apparently, is that the average ophthalmologist simply announces to the patient or to his family upon completion of the examination, that the patient's sight is beyond the help of medicine. The patient and his family are left to their own resources to find out what can be done to rehabilitate the patient to a useful life.

The social agency made the following suggestion, and I think it is an excellent one:

All ophthalmologists should acquaint themselves with the facilities available in their states to help re-educate and train the visually handicapped patient in skills that come within his potential.

Ophthalmologists also should equip themselves with the names and addresses of the various agencies that assist in the rehabilitation of the permanently visually handicapped person.

In other words, a positive approach to a permanent visual handicap would be better than the more common negative attitude. Instead of simply announcing to the patient that he is beyond any help, it would be better to tell him that we can offer no medical help but that we suggest he consult ———.

At the same time the patient would be given the names and addresses of the various agencies that have as their purpose the rehabilitation of the visually handicapped.

This simple procedure not only would soften the blow the patient feels when he learns he can expect no help from medicine, but also would give him a ray of hope that he can learn to lead a productive life and escape being a burden to society or family.

(Signed) William B. Clark,
New Orleans, Louisiana.

BOOK REVIEWS

Corneal Grafts. Edited by B. W. Rycroft. London, Butterworth & Co., Ltd., and St. Louis, C. V. Mosby Company, 1955. 285 pages, 155 illustrations, bibliography, index. Price: $13.50.

Through an oversight, a review of this excellent book, published in 1955, has not hitherto appeared in The Journal. My sincere apologies are here given to the author and his publishers for this serious error.

By coincidence this book appeared about six weeks after Townley Paton's *Keratoplasty* (Blakiston, New York) came out and the English-reading ophthalmic surgeons had a double treat set before them. Up to that time, the only books on the subject were those in French, *Les Greffes de la Cornée* by Paufique, Sourdille, and Offret (Masson et Cie, Paris, 1948) and *Les Greffes Lamellaires de la Cornée* by Rougier (Annequin, Lyon, 1950), both very good indeed.

Mr. Rycroft is chief of the Corneo-Plastic Unit and Eye-Bank, Queen Victoria Hos-

pital, East Grinstead, Sussex, which was established shortly after the end of World War II. An enormous amount of material has accumulated in the clinic and Rycroft's experience in keratoplasty assures us of authoritative work in this field.

There are 13 chapters, each written by an international authority that include Davson, Offret, Franceschetti and Maeder, Barraquer-Moner, Paufique, Castroviejo, the late G. Sourdille, Tudor-Thomas, F. Ridley, Amsler, Billingham, Maumenee and Paton, and three chapters by the editor. The entire field of the subject is beautiful covered by these experts.

Chapter 1, which discusses the fascinating history of the subject, by Rycroft, is noteworthy. It appears to be most complete. Davson gives us an excellent chapter on the anatomy and physiology of the cornea; Offret, the histopathology of the corneal graft; Franceschetti and Maeder, the indications for corneal transplants and selection of cases (particularly good).

Barraquer-Moner describes the techniques of full-thickness grafts; Paufique, lamellar keratoplasty (he was a pioneer in this phase); Castroviejo on keratectomies and retransplants (extraordinary good results); Sourdille on special methods; Tudor-Thomas on general complications; Ridley on the use of contact lenses in corneal grafts; Amsler on special features; Billingham and Rycroft on the preservation of the donor graft.

Maumenee describes his work on the biological problems and the malady of the graft; Paton on the legal aspects of the donor problem; and Rycroft concludes with chapters on special instruments (well illustrated) and results (obtained from an analysis of the literature, roughly ranging from 70 percent successes in favorable cases to 10 percent in unfavorable cases).

The editor has done a good job of co-ordinating the papers of the authors from various countries and of different languages, so that there is a pleasing uniform style of expression. Only those who have tried to do this know how difficult such a task can be. The printing is first class and the illustrations are uniformly good. The book is a fine addition to our growing library of the subject, and is highly recommended to all ophthalmic surgeons.

Derrick Vail.

PSYCHOSOMATIC OPHTHALMOLOGY. T. F. Schlaegel, Jr., M.D. Baltimore, Maryland, Williams & Wilkins Company, 1957. 523 pages. Price: $11.00.

Much thought, an enormous amount of reading, analysis, and abstracting of the literature, and the author's own considerable experience have gone into the preparation of this book. It should be a part of the personal library of every practicing ophthalmologist.

Dr. Schlaegel is assistant professor of ophthalmology at the Indiana University School of Medicine and, in addition, has had training in psychiatry. He has the right to speak with authority on his subject.

The literature on psychosomatic ophthalmology is widely scattered in the journals and textbooks of many medical disciplines over a long interval of time. The author has not been content merely to assemble and quote this material but has examined it with a critical eye and embellished it with many illustrative case reports and observations of his own.

Most physicians now realize that the incidence of disturbances of psychogenic origin or aggravation is very high in their daily practice but Schlaegel insists that the psychosomatic approach is necessary for proper treatment of *every* patient who walks into an ophthalmologist's office. He points out that every condition or disease is psychosomatic because both psychologic and somatic factors have a part in its course. He defines psychosomatic ophthalmology as a method of approach in which the somatic investigation is not neglected but in which a psychologic work-up is added so that psychologic and

somatic factors are studied in mutual interrelation.

Dr. Schlaegel's psychiatric training is evident in his facility with words. Not only is this volume a comprehensive reference work with a full bibliography but it also has a dynamic style which makes it absorbing reading throughout.

The book is divided into five parts dealing with the role of psychiatric factors in disease, general conditions involving the eyes about which psychologic factors are known, disturbances of function and structure of various parts of the eye, ocular conditions without specific anatomic location, and the psychosomatic approach to treatment.

While it is difficult to select specific portions for special commendation, I was particularly intrigued by the introductory chapters and those dealing with neurologic conditions, headaches, the extraocular muscles, the retina, and hysteria and malingering. The section on the psychosomatic approach to treatment, including excellent chapters on the blind and pharmacotherapy, is well worth the cost of the book.

It is to be expected that this work will arouse some controversy but, whether you agree with its philosophy or not, it should be read.

The publishers are to be complimented on the attractive format and binding.

David O. Harrington.

SURGERY OF HEAD AND NECK TUMORS. By Hayes Martin, M.D. New York, Hoeber-Harper, 1957. 424 pages, 599 illustrations, index. Price: $18.50.

Dr. Martin, associate professor of clinical surgery, Cornell University Medical College of New York, is an attending surgeon to the Memorial Hospital. He is a diplomate of the American Board of Radiology as well as the American Board of Surgery. He is widely known for his work in reconstructive surgery and surgery of malignancies of the head and neck. He has given us a good book on the subject.

The book is divided into two parts: Part I covers very neatly the basic principles of cancer surgery of the head and neck, a most difficult field; Part 2 consists of an atlas of operative procedures.

The illustrations are excellent line drawings in black and white, carefully executed and meticulously detailed, often step by step.

Nine pages are given to descriptions of operations for cancer of the skin of the eyelids. The procedures outlined are all well known and useful to most ophthalmologists. The descriptions and illustrations are adequate and give a chance for a quick comprehension of what it is all about.

This book will be particularly useful to our E.N.T. colleagues, who will find in it much of interest and value to them.

Derrick Vail.

ABSTRACT DEPARTMENT

EDITED BY DR. F. HERBERT HAESSLER

Abstracts are classified under the divisions listed below. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. Anatomy, embryology, and comparative ophthalmology
2. General pathology, bacteriology, immunology
3. Vegetative physiology, biochemistry, pharmacology, toxicology
4. Physiologic optics, refraction, color vision
5. Diagnosis and therapy
6. Ocular motility
7. Conjunctiva, cornea, sclera
8. Uvea, sympathetic disease, aqueous
9. Glaucoma and ocular tension
10. Crystalline lens
11. Retina and vitreous
12. Optic nerve and chiasm
13. Neuro-ophthalmology
14. Eyeball, orbit, sinuses
15. Eyelids, lacrimal apparatus
16. Tumors
17. Injuries
18. Systemic disease and parasites
19. Congenital deformities, heredity
20. Hygiene, sociology, education, and history

10
CRYSTALLINE LENS

Rodríguez Barrios, R. and Martínez Recalde, E. **Direct suction of congenital and juvenile cataracts.** Arch. oftal. Buenos Aires **32**:169-176, June, 1957.

Whether complete or incomplete, soft cataracts of whatever origin may be treated successfully in younger persons by means of direct aspiration through a modified Amsler cannula, attached to a double-barrelled syringe or to two paired common syringes. A much bevelled puncture is made at the limbus on the temporal side and a large, V-shaped discission is then performed with the sharp-pointed needle; suction, which is said to be easy, with one syringe is followed by irrigation with the other, saline-containing one, until the anterior chamber is deemed clear enough. The operation is claimed to produce little trauma and to minimize the risks of synechia formation, since all its four stages—paracentesis, rupture of the lens capsule, aspiration and irrigation—are carried out through a tiny, self-sealing wound. Added advantages are its simplicity, speedy performance and the possibility of its being repeated after a short interval. In 25 out of 28 cases the results were highly gratifying. In three, the opaque masses could not be adequately removed: one was a traumatic cataract in which inflammatory adhesions had already appeared, one a case of congenitally luxated lens where discission had previously been attempted, and one an intumescent cataract in a 30-year-old patient. (10 figures, 7 references)

A. Urrets-Zavalía, Jr.

Vannas, Salme. **Epithelial downgrowth into the anterior chamber and deep into the eye.** Acta. ophth. **35**:190-195, 1957.

The literature on the subject is briefly reviewed, and a case verified histologically is reported which followed a cataract extraction. This is the first case, histologically verified, reported in Finland. The epithelial invasion continued from the anterior chamber to the ciliary processes and to the anterior part of the retina. The etiology and prophylaxis of this disastrous complication is discussed and Vannes concludes that a Graefe knife incision and his triangle suture are effective prophylactic measures. Both procedures are a part of the routine technique of cataract extraction in Finland and Vannas believes that this accounts for the rarity of this complication in his country. (2 figures, 18 references) Ray K. Daily.

11
RETINA AND VITREOUS

Best, W. and Bohnen, K. **The alternation in the potential of the human electroretinogram in flickering light.** Acta ophth. **35**:273-278, 1957.

Reference is made to the authors' former report on the alternating character of the potential of the electroretinogram in a flickering light of 40 to 60 candle power. It was found that the positive potential of every other stimulus is smaller than that on either side of it. The objective of this investigation is to explain this phenomenon; 20 persons between 26 and 40 years of age were examined and in each the phenomenon was found constant. The alternation in the positive potential is due to the prolongation of every other off-effect; this prolongation is caused by the fact that the end of the second light stimulus falls in a phase of reduced excitability. The end of the third light stimulus again falls in a phase of normal excitability, because the previous response was incomplete. The alternation gives further support to the theory maintained by the author, that the alteration in the potential of the electroretinogram is related to the activity of the ganglion cells. The alternation in the potential is associated with subjective phenomena. The visual field is divided into two different areas; the periphery forms black rings similar to a Placedo disc, and the center remains uniform. In low flicker frequency the periphery appears blue and the center lemon yellow. (2 figures, 6 references).

Ray K. Daily.

Christiansson, John. **Changes in the vitreous in scurvy.** Acta ophth. **35**:336-360, 1957.

This is a detailed report of a laboratory investigation on guinea pigs in vivo. The experimental technique and methods of investigation are described in detail. The data comprise viscosity measurements, content of glucosamine, glucuronic acid, hydroxyproline and ascorbic acid of the vitreous and protein concentration in the aqueous humor. The summary shows that an ultrafiltrate of the vitreous humor of guinea pigs with experimental scurvy displays a lowered viscosity and a reduced concentration of glucosamine. Simultaneously there appears an increase of glucosamine in the aqueous. The factors giving rise to the reduction in viscosity of the vitreous have not been elucidated. The residual protein undergoes no quantitative changes. There appears to be an increase in the water content of the vitreous humor. The literature is reviewed and comparisons are made between the structure of the vitreous body and the ground substance of connective tissue. (3 figures, 14 tables, 50 references) Ray K. Daily.

Dorello, U. and Scorciarini-Coppola, A. **Results of surgical treatment of retinal detachment.** Rassegna ital. d'ottal. **26**:177-191, May-June, 1957.

The surgical results obtained in 400 cases of detachment of the retina were examined and the conclusion reached is that surgery should be performed whenever possible, since good functional results may be obtained in apparently hopeless cases. In 280 cases myopia varied from one to five diopters, in 79 from 5 to 10 diopters, and in 145 the myopia was greater than 10 diopters. Other causes of detachment were increased blood pressure, diabetes, familial factors and uremia. (3 figures, 4 tables, 27 references)

Eugene M. Blake.

Eisum, E. Frank. **Crater-like hole in the retina.** Acta ophth. **35**:200-203, 1957.

The author adds two more cases to the 80 reported in the literature. One patient, a woman, 60 years old, had a pit three diopters in depth in the right optic disc. Perimetry disclosed a central arcuate

scotoma with a horizontal lower border across the fixation area. The other patient, a man, 61 years old, had a pit in the lower temporal quadrant of the right eye. It is pointed out that sometimes these cases present a difficult differential diagnosis from glaucoma. The pathogenesis of these holes is discussed. (2 figures, 7 references) Ray K. Daily.

Gilje, K. and Nissen, A. J. **The early diagnosis of juvenile amaurotic idiocy. Lipoid lymphocyte degeneration.** Acta ophth. **35**:184-189, 1957.

The classification of the tapeto-retinal degeneration is briefly reviewed, and reference is made to the work of Bagh and Hortling who in 1948 demonstrated numerous lymphocytes containing vacuoles in the protoplasm in lipoidosis. In 1954 Nissen described 10 cases of juvenile amaurotic idiocy; in four of these vacuoles were found in the lymphocytes; in the other six cases this investigation was not made because this phenomenon has not been reported as yet. In this investigation the material comprised the 60 pupils of the school for the blind in Norway; among these 14 were diagnosed clinically as tapeto-retinal degeneration and six of these children had lipoid degeneration of the lymphocytes in the blood. These six had progressive loss of vision, increasing dementia, and four had epileptic attacks. The diagnosis of juvenile amaurotic idiocy was made in these six cases. The authors point out that examination of the blood is a valuable aid in the differential diagnosis of juvenile amaurotic idiocy at an early stage of the disease. (1 figure, 2 tables, 6 references) Ray K. Daily.

Grant, G. **External scleral buckling operation.** Arch. chil. de oftal. **14**:50-52, Jan.-June, 1957.

The author briefly summarizes the newer techniques used during the past several years for operation in retinal detachment. He feels than an external buckling operation, as described by Everett and Castroviejo is the most simple of all procedures and the least traumatic. His personal series is too small to permit an evaluation of its results. (2 references) Walter Mayer.

Houston, W. R. and Wise, G. N. **Circinate retinopathy. Part I.** A.M.A. Arch. Ophth. **58**:777-782, Dec., 1957.

A clinical diagnosis of Fuchs' retinitis circinata depends upon two factors: a definite macular lesion and a typical circinate figure emcompassing it. However, the literature indicates that the disease is not a specific entity. It is the authors' experience that the circinate figures seen in disciform macular degeneration, multiple miliary aneurysms with retinal degeneration, diabetic retinopathy, occasional lesions of Coats' disease, and some cases of venous obstruction have all been clinically identical or so similar as to indicate a common pathogenesis. (76 references) G. S. Tyner.

Houston, W. R. and Wise, G. N. **Circinate retinopathy. Part II.** A.M.A. Arch. Ophth. **58**:783-796, Dec., 1957.

The authors describe the lesions mentioned in the review of Part I. They conclude that circinate retinal lesions are manifestations of a local retinal response to hypoxia secondary to capillary or venous obstruction in the area. (10 figures, 68 references) G. S. Tyner.

Janert, H. **Diabetic retinitis proliferans.** Klin. Monatsbl. f. Augenh. **131**:633-640, 1957.

This is a histologic report on the eyes of a 51-year-old woman who died from vascular complications of diabetes. Glial and connective tissue proliferation were

found in the disorganized retina. Certain clinical signs speak for the probability that a proliferative retinitis will develop: an atrophic iris which transilluminates, irregular areas of increased retinal reflexes at the posterior pole, accumulation of aneurysms and cotton-wool patches. (4 figures, 5 references)

Frederick C. Blodi.

Kornerup. Tore. **Blood pressure and diabetic retinopathy.** Acta ophth. **35**:163-174, 1957.

The objective of this study was to correlate the types of retinopathy with changes in the general blood pressure. The author refers to his former study, and to his conception of a relative blood pressure, which is expressed as a percentage of the systolic and diastolic blood pressure in a nondiabetic of the same age. It was calculated from the data of insurance companies in Sweden and the continent. Material for this investigation consisted of 1,285 diabetics; their blood pressure was taken at rest in the morning, and they were classified according to the type of retinopathy. The tabulated data show the different types of retinopathy in relation to age, relative systolic blood pressure, and duration of the disease. These tabulated data show that in every group based on duration of the disease, the percentage of cases with blood pressure not exceeding normal decreases as the degree of retinopathy increases, and the frequency of cases with a relative systolic blood pressure greater than normal increases. In every group the percentage frequency of cases without retinopathy or with hemorrhagic retinopathy decreases with raised systolic blood pressure, and the percentage of the severe types of retinopathy increases. When the diastolic pressure is used as the criterion, the same general results are obtained, but the differences are less marked. The same conclusions emerged when classifying patients according to age. Of the patients with retinopathy 25 percent had a systolic blood pressure which did not exceed the blood pressure of healthy individuals of the same age. Of diabetics with a relatively normal blood pressure 35 percent had some type of retinopathy. The author concludes that diabetic retinopathy and a rise in blood pressure are closely related, parallel phenomena which are two distinct manifestations of the vascular disease typical of diabetes even of short duration, but that so far there is no basis on which to build a causal connection. (8 tables, 43 references) Ray K. Daily.

Kornerup, Tore. **Fundus hypertonicus and diabetic retinopathy.** Acta ophth. **35**: 175-183, 1957.

This is a contribution to the unsettled question of the interrelation between hypertension and diabetic retinopathy. The material consisted of the data on 1,353 diabetics which are classified according to Hanum's classification of the types of retinopathy, and to the degree of retinal hypertension according to Keith and Wagener. A hypertensive fundus was found in 33.7 percent of the patients. The frequency increased considerably with age, and to a lesser degree with duration. A hypertensive fundus is encountered in diabetics earlier than in nondiabetics. In higher age groups the incidence of a hypertensive fundus is about the same. The frequency of diabetic retinopathy was 46.2 percent. It increased markedly with the duration of the diabetes, but not with age. A hypertensive fundus was found in 40.3 percent of patients with diabetic retinopathy, and in 27.9 percent of those without it. The difference is not considered significant. A more striking difference between diabetic retinopathy and a hypertensive fundus appears in the table comparing their frequency distribution ac-

cording to age and duration of the disease. A hypertensive fundus was found in 6 percent of healthy patients under 40 years of age and in 35 percent of those over 40 years old. In diabetics the corresponding figures are 17 and 53 percent. With duration of diabetes there is some increase in the frequency of hypertensive fundi in the younger age groups, but it is not nearly as pronounced as with age. The hypertensive fundus appears to be a senile change aggravated by diabetes, and the two diseases appear to be entirely different entities. (5 diagrams, 2 tables, 50 references) Ray K. Daily.

Law, F. W. **The macular retinopathies.** Practitioner 178:534-539, May, 1957.

Anatomic factors, particularly the circulation in the choriocapillaris, fail to account for the vulnerability of the macula. Its high degree of differentiation and specialization offers the only explanation. The clinical picture, course, pathology, and treatment of the common macular retinopathies are discussed. (9 figures, 5 references) David Scher.

Lijo Pavia, J. **Central retinitis in one eye and complicated, luxated cataract in the other.** Rev. oto-neuro. oftal. 32:77-80, July-Sept., 1957.

The author describes a patient with a central retinitis in his left eye and a luxated complicated cataract in the right. The lens was luxated totally into the vitreous. He advised extraction of the cataractous lens and diathermy coagulation over the macular area in the left eye. He does not indicate the results of the treatment. (13 references) Walter Mayer.

Oksala, Arvo. **Changes in the vitreous after injection of air or saline solution.** Klin. Monatsbl. f. Augenh. 131:615-619, 1957.

Freshly enucleated eyes of steers were injected with 3 cc. of air or saline solution. A paracentesis preceded the injection. The eyes were then put in a refrigerator (−10°C) for 48 hours. The vitreous usually showed cavities filled with the injected material. In a few instances no cavity was found but only an area of coarse, granular degeneration. (4 figures, 9 references) Frederick C. Blodi.

Papst, W. and Heck, J. **The electroretinogram in circulatory disturbances of the retina and the influence of vasodilators, oxygen and hyperglycemia.** Klin. Monatsbl. f. Augenh. 131:598-610, 1957.

Nitroglycerol caused a constant but temporary increase in the b-wave in six patients with arteriolar sclerosis. More permanent results were obtained with Nepresol (Ciba). There was even improvement in a few cases of early retinitis pigmentosa. The administration of 96 percent oxygen for as long as 40 minutes did not influence the ERG in three patients with arteriolar sclerosis. Injection of glucose did not influence the ERG of normal patients, eyes with inflammatory or degenerative diseases, nor patients with occlusion of the central artery or vein. There was an increase in the b-wave after the injection in 13 out of 18 patients with arteriolar sclerosis. (6 figures, 4 tables, 33 references) Frederick C. Blodi.

Patz, Arnall. **The role of oxygen in retrolental fibroplasia.** Sinai Hosp. J. 6: 3-22, April, 1957.

In this address the history of the implications of oxygen therapy is reviewed. In animal experiments the characteristic changes of early human retrolental fibroplasia were reproduced: endothelial nodules in the nerve fiber layer; budding of capillaries into the vitreous with tuft formation; retinal edema, retinal and vitreous hemorrhages; vitreous degeneration. The retinal vessels do not reach the

anterior retina until the eighth month of gestation, so a child born at seven months has no circulation there. This area is very susceptible to oxygen damage. Oxygen initially produces vasoconstriction and suppression, but later produces proliferation. Constriction lasting over two days can produce obliteration. Metabolism therefore is affected. This secondary proliferative effect may occur under sustained over-oxygenation, or after withdrawal from oxygen when the vessels are still closed.

The chief factor in oxygen damage is duration of exposure. The severity of the lesions is also directly proportional to the oxygen concentration. Immaturity also increases susceptibility, and continuous exposure is more dangerous than intermittent.

A list of recommendations for nurseries is given. (12 figures, 2 charts, 1 table)

Harry Horwich.

Pau, Hans. **Asteroid hyalitis.** Klin. Monatsbl. f. Augenh. **131**:610-615, 1957.

The globe of a 55-year-old man with melanoma of the choroid and scintillation of the vitreous was examined histologically. There was proliferation of the pigmented ciliary epithelium and the author believes that these cells form the deposits in the vitreous. They become smaller with time and finally disintegrate. (6 figures, 13 references)

Frederick C. Blodi.

Rodger, F. C. **Posterior degenerative lesion of onchocerciasis.** Brit. J. Ophth. **42**:21-37, Jan., 1958.

The inflammatory lesions are described as separate from those due to degenerative changes in onchocerciasis. It seems probable that vitamin A is a factor in the production of degeneration when combined with a toxin liberated by the worms. (1 figure, 3 tables, 31 references)

Lawrence L. Garner.

Sauter, H. **Centennial: "embolism of the central retinal artery."** Med. Klin. **52**: 1620-1624, Sept. 13, 1957.

The first diagnosis of "embolism of the central retinal artery" was made 100 years ago by A. v. Graefe. As an etiologic factor of the disease a real embolus vary rarely is found and in the majority of the cases examined the changes of endarteritis were found. The original treatment of paracentesis and iridectomy not only did not cause a vascular dilation but, according to animal experiments, the arterioles of the retina respond with a contraction. Believing that a spasm is the cause of the occlusion, medical therapy with vasodilators should be considered for a long time, improvements have been observed to start after many weeks of treatment.

Stephen G. Seech.

Schepens, C. L., Okamura, I. D. and Brockhurst, R. J. **The scleral buckling procedures.** A.M.A. Arch. Ophth. **58**:797-811, Dec., 1957.

This very valuable paper outlines the authors' technique for the various forms of scleral buckling using polyethylene tubing to indent the area of sclera and choroid. They believe this method to be safer and more apt to produce reattachment than either the typical multiple diathermy puncture retinopexy or lamellar resection operations. Furthermore, early ambulation is permitted and reoperations, if they should become necessary, are easier. (6 figures, 11 references)

G. S. Tyner.

Vannas, S. and Orma, H. **Experience of treating retinal venous occlusion with anticoagulant and antisclerosis therapy.** A.M.A. Arch. Ophth. **58**:812-828, Dec., 1957.

In this paper from Finland the authors report the treatment of 75 patients with retinal venous occlusions; 37 were treated

with anticoagulants and 36 were used as controls. The results achieved were as follows (control group in parentheses): good in 59 percent (14 percent), fair in 20 percent (28 percent), poor in 13 percent (16 percent), and nil in 8 percent (42 percent). They believe that even short periods of treatment are sometimes efficacious, but the more prolonged regime is recommended. (4 tables, 29 references, and a detailed appendix report on each case) G. S. Tyner.

van Walbeek, K. **Anatomico-pathologic examination of a lamellar scleral resection.** Ann. d'ocul. 190:698-702, Sept., 1957.

This histologic report is on an eye which underwent two lamellar scleral resections two months apart. Two months after the second resection the patient died and the eye was enucleated. On sectioning the eye, the author notes that the first resection (four months old) had flattened out but the retina remained in contact with the choroid and sclera. The more recent resection was still in place and pieces of nylon suture material were still visible. One can conclude that a scleral fold lasts from two to four months. (4 figures, 4 references) David Schoch.

Wadsworth, Joseph A. C. **The vitreous.** A.M.A. Arch. Ophth. 58:725-734, Nov., 1957.

In a well illustrated article, the author discusses the role of the vitreous in retinal detachment. He points out that shrinkage and detachment of the vitreous is an extremely common occurrence in people over 50 years of age. In most cases no damage results. However, if in the course of shrinkage there is an adhesion of the vitreous to the retina, subsequent shrinkage of the vitreous may result in a retinal tear as a result of traction. This may occur after choroiditis. The vitreous may form strands which tear off the retina or retinochoroidal adhesions may be so dense as to prevent retinal separation.

Primary horseshoe-shaped tears are probably formed by an adhesion between retina and vitreous in an eye in which the vitreous has been shrunk. Secondary holes may be caused by small localized areas of old chorioretinitis. When the fluid migrates to the lower portion of the globe, sufficient pushing force is exerted on the localized adhesions to form a hole. (42 figures, 43 color plates, 36 references) G. S. Tyner.

Weizenblatt, Sprinza. **Differential diagnostic difficulties in typical retinoblastoma.** A.M.A. Arch. Ophth. 58:699-709, Nov., 1958.

An eight-year-old boy with retinoblastoma presented the following unusual signs; recurrent inflammation of the globe associated with large, ring-shaped granular corneal deposits; a change in color of the iris from hazel to blue; normal pupillary reaction; normal ocular tension until late when glaucoma developed as a result of a cataract; swollen disc with diffuse tumor infiltration of the disc and the surrounding retina, but no discrete tumor mass. Autopsy showed direct extension of the tumor along the nerve into the brain. Involvement of the other optic nerve was also discovered at autopsy. (12 figures, 7 references) G. S. Tyner.

Wise, George N. **Coats' disease.** A.M.A. Arch. Ophth. 58:735-746, Nov., 1957.

The author discusses and explains the various clinical and microscopic findings in a variety of conditions—all considered by some as Coats' disease and by others as separate entities. The diseases discussed are Leber's exudative retinitis and multiple miliary aneurysms, Von Hippel's disease, circinate retinopathy, Reese's massive retinal fibrosis and some cases of retrolental fibroplasia. (9 figures, 37 references) G. S. Tyner.

Wudka, E. and Leopold, I. H. **Experimental studies of the choroidal vessels.** A.M.A. Arch. Ophth. **58**:829-849, Dec., 1957.

The present study concerns itself with the reaction of the choroid to physical agents used in retinal detachment surgery, namely, mechanical agents such as scleral folding and thermal agents such as diathermy. Lamellar scleral resections produced only a temporary alteration of the choroidal circulation. After diathermy coagulation there is a tendency for the lesions to become revascularized to compensate for the changes produced. (9 figures and 248 references) G. S. Tyner.

12

OPTIC NERVE AND CHIASM

Bialasiewicz, A. **Damage to the optic nerve through endogenous medicines in Indonesia.** Klin. Monatsbl. f. Augenh. **131**:797-799, 1957.

Six cases of partial or total blindness are reported that followed the drinking of an unknown solution prepared by a local medicine man. Frederick C. Blodi

Haberland, Catherine. **Bilateral optic nerve glioma with misleading encephalogram.** Psychiatria et Neurologia **134**:215-223, Sept.-Oct., 1957.

The author reports a case of bilateral glioma of the optic nerve in which the diagnosis was made difficult by partial coalescence of the thalamus which prevented filling of the third ventricle for encephalography. The occurrence of café-au lait spots in the skin may direct attention to tumor of the optic nerve in a patient with bilateral signs of ocular disease. (3 figures, 10 references)

Irwin E. Gaynon.

Levatin, P. **Increased intracranial pressure without papilledema.** A.M.A. Arch. Ophth. **58**:683-688, Nov., 1957.

A typical Foster Kennedy syndrome in a 43-year-old woman who had a left parasagittal meningioma is reported. A 12-year old-boy is described who showed an elevated spinal fluid pressure but no papilledema. Both discs appeared somewhat atrophic. At surgery a massive glioma was discovered which affected the intracranial portion of both optic nerves. This lesion brought about optic atrophy but had mechanically prevented the development of edema of the nerve heads. (1 figure, 10 references) G. S. Tyner.

Liss, L. and Wolter, J. R. **The histology of the glioma of the optic nerve.** A.M.A. Arch. Ophth. **58**:689-694, Nov., 1957.

A tumor of the optic nerve in a four and one-half year-old girl is described. The cellular elements of the tumor consisted of elevated spindle-shaped bipolar cells, or Schwann elements; round multipolar oligodendroglia; and astrocytes or astroblasts. There was characteristic invasion of the subdural space and the neoplasm classed as an oligodendroglioma. (17 figures, 13 references) G. S. Tyner.

Misar, Rainer. **The final results of retrobulbar neuritis.** Klin. Monatsbl. f. Augenh. **131**:771-776, 1957.

In five patients the disappearance of the central scotomas was carefully studied. It was interesting to note that for a certain target and illumination the scotoma will suddenly disappear without the patients noticing an improvement in vision. The next smaller target, however, is at that stage not yet perceptible on the Goldman perimeter. (17 figures, 5 references) Frederick C. Blodi.

Pasino, Luigi. **Avulsion of the optic nerve.** Rassegna ital. d'ottal. **26**:111-117, March-April, 1957.

A 40-year-old man fell, striking his head violently against the door handle, with a

resultant evulsion of the right eyeball. The eyeball was completely outside the orbit and all of the extraocular muscles were torn away from the globe. There was loss of cerebrospinal fluid, followed by hemianopsia of the remaining eye as a result of stretching the chiasm, and occipital headache. (3 figures, 7 references) Eugene M. Blake.

Peterson, Hans Peter. **Colloid bodies with defects in the field of vision.** Acta ophth. 35:243-272, 1957.

The literature is briefly reviewed and 30 cases, which were collected over a period of 10 years, are reported in detail with drawings of the disc and visual fields. Most of the patients had been examined repeatedly; in seven the colloid bodies had grown in size and increased in number and in others they remained unchanged. A characteristic feature is that in all cases the physiologic cupping of the optic disc was absent. The majority of the patients had visual field defects in the form of a Bjerrum scotoma or peripheral contractions or both. Superficially situated bodies do not produce significant visual field defects, but deeply imbedded bodies may affect the visual field significantly. Deeply situated bodies are at times difficult to differentiate from a choked disc. Colloid bodies usually are not accompanied by hemorrhage or compression of the veins. The final diagnosis may not be made until the colloid bodies have broken through to the surface of the disc. In all cases of protuberance or fullness of the optic disc of unknown etiology the possibility of colloid bodies should be kept in mind. A grayish black coloration or an opalescence deep in the disc, described as marbling, is an indication of the presence of deeply imbedded colloid bodies. To determine whether colloid bodies may be a consequence of some previous disease of the optic nerve the author examined 110 patients who had recovered from a lesion of the optic nerve; in none of them were colloid bodies or marbling of the disc found. There seems to be no connection between colloid bodies around the macula and colloid bodies on the disc. Of 24 patients with colloid bodies around the macula only one also had colloid bodies on the disc. On the other hand, a relationship between tapeto-retinal degeneration and colloid bodies on the disc seems probable; of 47 patients with pigmentary degeneration of the macula four were found to have colloid bodies on the disc. Two patients in the author's series of 30 cases had pigmentary retinal degeneration. (57 figures, 23 references) Ray K. Daily.

Slade, H. W. and Weekley, R. D. **Diastasis of the optic nerve.** J. Neurosurg. 14: 571-574, Sept., 1957.

A case is reported of diastasis of the right optic nerve caused by an abnormal branch of the right internal carotid artery. The postmortem examination showed this abnormal branch going through the right optic nerve producing a diastasis 5 mm. long. Death was caused by a massive hemorrhage into the middle cranial fossa. (4 figures, 4 references)

Ernest E. Hessing.

13

NEURO-OPHTHALMOLOGY

Fowler, F. D. and Matson, D. D. **Gliomas of the optic pathways in childhood.** J. Neurosurg. 14:515-528, Sept., 1957.

Thirteen verified cases of optic pathway gliomas in children are presented. They are divided into two distinct groups, the intraorbital of which there were four and the intracranial, numbering nine. The average age for the whole group was four and one-half years. Proptosis and papilledema in the affected eye were present

in all four of the intraorbital group, whereas diminished visual acuity was the prominent symptom in the intracranial group. A diagnosis of von Recklinghausen's disease was made in four of the nine in the intracranial group. Radiographic evidence of enlargement of the optic foramen and erosion of the sella turcica are important diagnostic signs. Plain roentgenograms of the skull were abnormal in all nine of the children with intracranial lesions.

The authors are strongly in favor of a transfrontal exposure of the roof of the orbit and the region of the chiasm. One cannot be sure before operation whether the tumor is completely intraorbital. With the anterior approach the optic foramen cannot be exposed adequately and therefore an extension through the foramen cannot be seen.

Of the 13 patients, all treated surgically, all four of the intraorbital group are alive (and have good vision in one eye) eight years, five years, eight months and one month after operation; and of the remaining seven children three have been well for four years, two for two years, one for six months and one for one month. (7 figures, 35 references) Gerald S. Ryan.

Leinfelder, P. J. **Various syndromes associated with visual field changes.** Iowas St. Med. Soc. **47**:628-630, Oct., 1957.

The author discusses the causes of central scotoma, temporal anopsia, and homonymous hemianopsia and describes the visual field defects caused by lesions of the occipital lobe.

Edward U. Murphy.

Lyle, Donald J. **Neuro-ophthalmology.** A.M.A. Arch. Ophth. **58**:758-774, Nov., 1957.

The year's literature is abstracted and reviewed. (177 references) G. S. Tyner.

Palumbo, L. T. **A new concept of the sympathetic pathways to the eye: a new technique to avoid a Horner's syndrome.** Surgery **42**:740-748, Oct., 1957.

The author disputes the usual description of the sympathetic pupillociliary pathways controlling the dilation of the pupil in adult man. According to previously accepted description, removal of the lower part of the stellate ganglion or resection of the first or second thoracic rami, or both would produce a Horner's syndrome.

Palumbo postulates that the preganglionic neurones controlling the pupil enter the upper portion of the stellate ganglion by a separate paravertebral route. They leave the ventral roots of the eighth cervical and the first and second thoracic nerves, and he refutes the concept that these pathways pass via the first ramus communicans to the first thoracic ganglion. To substantiate his concept he cites 45 operations on the upper thoracic sympathetic chain, done via an anterior transthoracic, transpleural incision, in which not a single case of Horner's syndrome occurred. (6 figures, 26 references)

Irvin S. Pilger.

Pietruschka, G. and Stump, W. **A case of rare pupillary disturbance.** Arch f. Ophth. **159**:420-432, 1957.

The authors describe the combination of several paradoxic pupillary phenomena in a woman, aged 56 years, with neurosyphilis. She had bilateral paradoxic convergence reaction and also bilateral paradoxic abduction and adduction movements with pupils which did not react to light. A search through the literature revealed no similar case of combined paradoxic pupillary movements. The authors describe the details of studies of the patient and discuss the genesis of the disturbance at length. (44 references)

F. H. Haessler.

Ricci, A. and Werner, A. **Neurosurgical verification of the role of the internal carotid in certain chiasma syndromes.** Schweiz. med. Wchschr. 87:1190-1194, Sept. 21, 1957.

Two cases of chiasmal syndrome with verified compression of the optic nerve by the internal carotid artery are reported. One patient had an incomplete binasal hemianopsia; decreased visual acuity and enlargement and calcification of the internal carotids; the other had bitemporal hemianopic scotoma, decreased visual acuity and the optic nerve was found to be wedged against the roof of the optic canal. Removal of the roof of the optic canal gave functional recovery in each. (6 figures, 22 references)

Irwin E. Gaynon.

Rintelen, F. and Leuenberger, A. **The differential diagnosis and treatment of craniopharyngeoma.** Schweiz. med. Wchschr. 87:1189-1190, Sept. 21, 1957.

The pathology, clinical findings, the importance of an exact visual field, the differential diagnosis and the treatment (with its 20 to 40 percent operative mortality) in a 64-year-old woman with craniopharyngioma are discussed. (2 figures, 9 references) Irwin E. Gaynon.

Sternberg, A. and Véli, M. **Divergentia paralysis on the basis of three cases.** Wzemeszet 2:71-75, 1957.

Three patients had divergence paralysis, apparently resulting from encephalitis. One of the patients noted temporary fourfold vision as a result of a cortical lesion. The clinical pattern, diagnosis, etiology and prognosis of divergence paralysis are discussed. Gyula Lugossy.

Warrington, E. and Zangwell, O. L. **Study of dyslexia.** J. Neurol. & Psychiat. 20:208-215, Aug., 1957.

The syndrome of "pure alexia" (word blindness; inability to read due to a central lesion) is probably invariably associated with other defects such as right hemianopia, an apparently specific defect of color vision, and minor grades of dysphasia and dysgraphia, and is associated with a lesion of the dominant occipital lobe. The case of a left occipitoparietal meningioma in a 41 year old, intelligent, right handed man is presented in detail. Word-blindness was his major symptom and persisted following remedial surgery. Photographic eye movement records showed gross abnormality which was attributed to disordered perception, with apparent inability to compensate for his right hemianopia. (3 figures, 21 references) Joseph L. Dowling, Jr.

14

EYEBALL, ORBIT, SINUSES

Bamert, W. **The pathology of secondary tumors of the orbit in early childhood.** Schweiz. med. Wchschr. 87:1201-1202, Sept. 21, 1957.

One case of unilateral exophthalmus and one of bilateral exophthalmus, both occurring in early childhood, and each secondary to a neuroblastoma, are reported. (2 figures, 7 references)

Irwin E. Gaynon.

Barthelmess, G. **Coordimetry in displacements of the globe.** Klin. Monatsbl. f. Augenh. 131:662-669, 1957.

Decreased or impaired motility of the eye due to orbital lesions can be mapped accurately on the Hess screen. Orbital tumors will influence ocular motility according to their location. The course of the disease can also be followed quantitatively. Fractures of the orbital bones can be evaluated. (5 figures, 16 references)

Frederick C. Blodi.

Bonnet, Paul. **Exenteration of the orbit: a life-saving operation.** Arch. d'opht. 17: 470-472, 1957.

The author describes the operation of orbital exenteration and reports his more than 50 cases. He considers it an excellent operation with excellent results. He describes in detail the results of exenteration in two women with recurrent conjunctival melanoma and stresses the life-saving aspect of the procedure.

P. Thygeson.

Khramelashvili, N. **Exophthalmos caused by a tumor of the cheek.** Vestnik oftal. 6:41-42, Nov.-Dec., 1957.

A woman, 55 years old, had exophthalmos with displacement of the left eye down and out, and diplopia of ten days duration. A tumor was palpated at the inner upper wall of the orbit which was adherent to the eyeball. The media and fundus were negative. There was a nodular mobile tumor on the left cheek, not adherent to the skin, 5 by 3 cm. in size. It was first noticed 10 years previously and it increased in size gradually. The test of the blood and X-ray studies of the orbit and the sinuses were negative. The tumor of the cheek was removed in its capsule and the pathologic diagnosis evaluated for the consequent removal of the orbital tumor. Three weeks after the operation the exophthalmos, diplopia and chemosis of the conjunctiva disappeared and the mobility of the eye became normal. The exophthalmos and other ocular abnormalities were probably the result of blocking of the orbital veins.

Olga Sitchevska.

MacCarty, C. S., Lillie, J. C., Daly, D. D., Hollenhorst, R. W. and Holman, C. B. **Blindness secondary to unusual cyst of the sphenoid sinus.** A.M.A. Arch. Ophth. 58:747-750, Nov., 1957.

An unusual case is presented in which a patient became blind from an apparently inoperable cyst of the sphenoid sinus. At operation a benign cyst was found and eradicated with return of vision. (5 figures, 1 reference) G. S. Tyner.

Maksimenki, I. **Osteoma of the orbit originating from the frontal sinus.** Vestnik oftal. 6:37-38, Nov.-Dec., 1957.

A woman, aged 30 years, noted exophthalmos 10 days before her hospital admission. The left eye was displaced down and in and its motion outward and upward was limited. The media, fundus and vision were normal. A hard, immobile, smooth tumor was palpated between the eyeball and the orbital wall. The X-ray study showed a compact tumor in the frontal sinus. Excision proved difficult, as the tumor was hard and smooth, and a chisel had to be used for removal of the tumor in small fragments. A resection of the lateral margin of the orbit after Kroenlein had to be done as the tumor extended deep into the sinus towards the cranium. The postoperative course was uneventful. The exophthalmos disappeared, the vision remained normal, and normal motility of the eyeball was restored. The pathologic examination showed a bony tumor, compact in some places, spongy in others.

This was the only case of an osteoma seen in this hospital among 72,000 patients during the last nine years.

Olga Sitchevska.

Marin-Amat, M. **Capsuloscleral implants.** Arch. Soc. oftal. hispano-am. 17: 625-628, June, 1957.

The author designates the new Arruga implant as capsuloscleral. He discusses the relation of this implant to the anatomy and physiology of the orbit, and concludes that it has no advantage over the intra-orbital implant. He holds that only an intrascleral implant, or the Mules operation, is in accord with the anatomy and physiology of the orbit. Ray K. Daily.

Nirankari, M. S., Singh, M., Manchanda, S. S., Chitkara, N. L. and Maudgal, M. C. **Eosinophilic granuloma of the orbit.** A.M.A. Arch. Ophth. **58**:857-861, Dec., 1957.

A seven-year-old boy with eosinophilic granuloma of skull and orbit had exophthalmos. The scapula was also involved. Treatment by curettage gave some relief. (8 figures, 8 references) G. S. Tyner.

Osorio, Luis A. **Primary tumors of the orbit.** Arq. brasil. de oftal. **20**:75-123, 1957.

From 1934 to 1956 twelve primary tumors of the orbit were observed in patients from two to 68 years of age. Meningioma, hemangioma, angiosarcoma, primary cholesteatoma, spongioblastoma of the optic nerve, neurilemmoma (neurofibroma), and mixed tumor of the lacrimal gland were encountered. The treatment in these cases consisted of radiation, antibiotics, antiluetic therapy, exenteration of the orbit, and removal of the tumor by the approaches of Dandy, Knapp and Lagrange, and Kroenlein. Primary tumors of the orbit present a difficult diagnostic problem. Classification is difficult because of their pleomorphism. Exploration of the orbit is most satisfactorily done by the transpalpebroconjunctival approach of Knapp and Lagrange, which results in a minimum of trauma and disfigurement. Complete blood studies are recommended in all cases of unilateral exophthalmos without signs of inflammation. A lymphoblastoma may be the underlying neoplasm. (28 figures, 25 references) James W. Brennan.

Schneider, O. **Orbital implants following the technique of Arruga-Moura.** Arch. chil. de oftal. **14**:24-28, Jan.-June, 1957.

The author describes his method of implanting an Arruga type of acrylic implant after an enucleation. This implant has two tongues which may erode through the conjunctiva, usually between the eighth and fortieth day.

He summarizes his findings in 22 cases of enucleation with this type of implant, three of which he had to remove because they were not tolerated. (1 table, 3 references) Walter Mayer.

Smith, Byron. **Diplopia in depressed orbital fractures.** Plastic and Reconst. Surg. **20**:318-322, Oct., 1957.

Methods of diagnosing and measuring diplopia and defects of muscle are enumerated and described. The three types of diplopia (physiologic, monocular and binocular) are differentiated. Methods of treatment of orbital fracture and diplopia are presented. If there is a complicating disease of the lacrimal sac, this should be repaired first. All bone work is performed before any extraocular muscle surgery is done. If diplopia exists after bony reconstruction has been completed, conservative treatment is indicated for six months. If it is not possible to give single binocular vision in all fields, an attempt should be made to move the binocular field which is present into a central position. Alston Callahan.

Vancea, P. **Mucocele of the maxillary sinus with orbital evolution.** Arch. d'opht. **17**:465-469, 1957.

The author reports a case of maxillary sinus mucocele with orbital involvement resulting in upward and inward displacement of the globe with disturbance in motility and drop in vision to 1/10. Cure was obtained by extirpation of the mucocele and partial resection of the orbital wall of the maxillary sinus. After a review of the literature the author concludes that mucocele of the maxillary sinus is a well-defined entity which can evolve slowly. Histologic examination permits differentiation from paradental cyst. The differential diagnosis from neoplasms originat-

ing in the nose is detailed. (2 figures)
P. Thygeson.

Vannini, A. and Pettinati, S. **Clinical and radiological considerations in congenital anophthalmos.** Rassegna ital. d'ottal. 26:214-225, May-June, 1957.

While there is always a very small eyeball in congenital anophthalmos the latter is often so small as to be unidentifiable by palpation. In more than one-half of the cases the defect is bilateral. At times the only evidence of the presence of a microphthalmic rudiment is the synchronous movement of the stump when the existing eye is rotated. Three stages of this defect are recognized, first when development is arrested in its earliest stage, secondly, when the arrest of development occurs later, and thirdly, the degenerative form. X-ray pictures and injection of the angular vein with an opaque solution and the electroencephalogram are aids in the study of congenital anophthalmos. (8 figures, 5 references) Eugene M. Blake.

Vannini, A. and Pettinati, S. **Simultaneous radiographic study of the orbital veins and tomography.** Rassegna ital. d'ottal 26:81-90, March-April, 1957.

Opacification of the orbital veins and the cavernous sinus obtained by the introduction of contrast media through the angular facial vein, aids greatly in diagnostic precision. The method permits the elimination of several possible causes of error. The author's method and experience are reported. (6 figures, 29 references) Eugene M. Blake.

15

EYELIDS, LACRIMAL APPARATUS

Belmonte, Jose. **The action of antibiotics in dacryocystitis.** Arch. Soc. oftal. hispano-am. 17:705-708, July, 1957.

Belmonte reviews the pathology of dacryocystitis, and describes the effect of antibiotics on the pathologic process. The distension of the lacrimal sac and the infiltration of the mucous membrane with leucocytes and plasma cells in dacryocystitis disappears under the action of antibiotics. The mucous membrane atrophies, the infiltration is diminished and the contraction of the increased amount of fibrous tissue leads to a shrinking of the sac and a narrowing of its lumen. This effect of antibiotics is held responsible by the author for the increased percentage of failures in dacryocystorhinostomy. Antibiotics may be used when it is essential to extirpate the lacrimal sac, for example in corneal ulcer or as a preparation for intraocular surgery, but when dacryocystorhinostomy is contemplated antibiotics should be avoided and the lacrimal sac be permitted to dilate and hypertrophy. (2 figures) Ray K. Daily.

Gáll, J. **Alterations of lacrimal ducts in trachoma.** Szemeszet 2:91-94, 1957.

In pronounced trachomatous alterations of the conjunctiva the X-ray examination of the lacrimal ducts filled with radioopaque substance revealed anatomic change in 68 percent of the patients who had no pertinent complaints and apparently permeable ducts. Gyula Lugossy.

Geiler, Gottfried. **The biorheutic orthology and pathology of the lacrimal gland.** Arch. f. Ophth. 159:371-383, 1957.

Only an exact knowledge of the biorheutic orthology permits an exact evaluation of individual observation and makes a possible differentiation of a senile physiologic finding from pathologic process. The author applies this abstract concept to observations of sections of the lacrimal gland and particularly to his studies of the pathogenesis of Sjøgren's syndrome. He analyzes particularly the difficult distinction between chronic inflammation of

normal lymphadenoid tissue and leukemic infiltration, which is of differential diagnostic importance. (7 figures, 27 references) F. H. Haessler.

Llopis Rey, Juan J. **Calcium crystals included in the giant cells of a chalazion.** Arch. Soc. oftal. hispano-am. 17:449-455, May, 1957.

This is a detailed description of the frozen microscopic sections of a chalazion in which calcium crystals were found within the giant cells. In the tissue imbedded in paraffin these crystals could not be demonstrated. The author believes that the calcium crystals are formed about coagulated extracellular proteins, and are subsequently included within the giant cells by phagocytic action. This conclusion is based on the extracellular presence of calcium granules, on the phagocytic activity demonstrated in the sections, and on the presence of giant cells about these crystals even after they have been included in one of the cells. (5 photomicrographs, 8 references) Ray K. Daily.

Nemeth, L. **The operation for paralytic ectropium.** Klin. Monatsbl. f Augenh. 131:815-819, 1957.

In patients with permanent facial palsy the orbicularis muscle is shortened with a suture. The skin incisions are made from the internal canthus toward the middle of the lower lid. The muscle suture is placed through the orbicularis at the middle of the lower lid and anchored at the internal canthal ligament. (5 figures, 5 references) Frederick C. Blodi.

Radnót, M. **Restoration of lacrimal ducts.** Szemeszet 2:84-89, 1957.

On the basis of observations made in 300 operations for the restoration of the lacrimal passages, the author is of the opinion that any tearing that occurs in cases of subacute or chronic dacryocystitis, ectasia of the lacrimal sac, stenosis of the nasolacrimal duct, or after the removal of the lacrimal sac, indicates that the lacrimal pathways should be restored. If possible, dacryocystorhinostomy should be accomplished. If this is impossible, canaliculorhinostomy is suggested. The method of the latter operation devised by the author was accomplished with success in all fourteen cases. These operations represent for the patient a trauma hardly exceeding that resulting from the removal of the lacrimal sac. The prognosis is rather good in childhood also. Gyula Lugossy.

Segal, N. **Late results after transplantation of Stensen's duct.** Ophthalmologia 2: 78-81, Jan.-March, 1957.

The postoperative course over a period of three years after transplantation of Stensen's duct is related. As compared to early results, changes are found which raise the problem of the durability of results and of the qualitative changes of parotid secretion. An increase of proteinemia is found as well as the appearance in the secretion of glucose and sodium chloride in concentrations similar to those of the lacrimal secretion (glucose and sodium chloride are normally absent from parotid secretion).

Biomicroscopic examination disclosed small, peripheral plates of xerosis which do not progress and do not impair visual acuity.

The conclusions indicate that if the transplantation of Stensen's duct does not cure xerophthalmia, it leads, nevertheless, to its improvement and prevents the onset of new foci, by permanently bathing the eye. Despite all its drawbacks, the transplantation of Stensen's duct may be considered as the most efficient among the methods which have been advocated and can be recommended in all cases of xerophthalmia. Author's summary.

Sherman, A. E. **Reconstruction of the upper eyelids.** Plastic and Reconst. Surg. 20:323-327, Oct., 1957.

The author presents seven cases (15 photographs) to illustrate some of the basic procedures used for reconstruction of the upper lid. Cicatricial ectropion resulting from burns requires excision of the scar tissue, two intermarginal lid adhesions, and a free skin graft to fill the defect, according to the technique perfected by Wheeler. The Esser epidermal inlay and the Gillies epithelial outlay methods of grafting skin to eyelids are deforming and can endanger the cornea because of temporary increase in the ectropion in the early postoperative period. Full thickness loss of one-fourth of the upper eyelid due to congenital coloboma, excision for tumors, or resulting from other trauma, can be very satisfactorily repaired by means of the Wheeler halving repair. For a full thickness loss of half of the upper eyelid, adequate repair can be obtained by Wheeler's method of using the Celsus-Knapp type sliding flap from the temporal region. The incisions from the upper part of the defect and from the lateral canthus must diverge and extend well into the temporal region. The Hughes lower lid reconstruction is quite satisfactory when applied to the upper lid. Callahan reported the use of a free composite graft from the opposite upper lid to partially fill a surgical loss of the mid half of the tarsal portion of the upper lid. When pedicle flaps are used, the eyelid portion should be as thin as possible and a minimal amount of new scarring produced. Alston Callahan.

16
TUMORS

Bech, Knud. **Malignant superciliary lymphoma.** Acta ophth. 35:204-209, 1957.

A fatal case of this disease is reported in a girl, 18 months of age. The lymphoma first appeared in the left eyebrow. Subsequently tumors appeared on the face, and finally the lesion involved the internal viscera. The literature on the histological classification, prognosis and therapy is reviewed. (3 figures, 10 references) Ray K. Daily.

Kleinert, Heinz. **Avulsion of the globe with hemianopia of the other eye.** Klin. Monatsbl. f. Augenh. 131:823-827, 1957.

Traumatic avulsion of an eye with only little damage to the orbital bones occurred in a 46-year-old construction worker. The globe was practically intact and attached to 55 mm. of optic nerve. The hemianopia in the other eye was probably caused by direct damage to the chiasm. (4 figures, 13 references) Frederick C. Blodi.

Nover, A. and Zielinski, H. W. **Differential diagnosis of orbital tumors and tumors of the optic nerve.** Klin. Monatsbl. f. Augenh. 131:577-598, 1957.

This study is based on 30 orbital tumors and 25 tumors of the optic nerve. Exophthalmus was present in all but four patients. Lateral displacement of the globe occurred only with orbital tumors and so did retinal folds and edema. The patients with tumor of the optic nerve showed either optic atrophy (17 cases) or papilledema (8 cases). Disturbances of motility were present in 22 patients with orbital tumors and in only one patient with a tumor of the optic nerve. Proptosis was the usual first sign of orbital tumor. Frequently the first symptom in patients with tumor of the optic nerve was decreased vision. Exophthalmometry and X-ray examination were the most useful methods of examination. Angiography was valuable in a number of patients. It may show displacement of the ophthalmic artery or demonstrate the vessels of the tumor.

By far the most frequent tumor of the optic nerve was the glioma. The commonest orbital tumors were (in order of frequency): sarcoma, metastatic tumor, angioma and secondary carcinoma. All tumors of the optic nerve were excised in two stages. First, the intracranial part of the nerve was removed and later the orbital part. The approach to the orbital tumors varied. (11 figures, 4 tables, 81 references) Frederick C. Blodi.

17

INJURIES

Mazur, Janina. **Intraocular foreign bodies.** Klinika Oczna **27**:293-298, 1957.

The author made a statistical study of 109 eyes with intraocular foreign bodies treated in the Krakow Medical Academy Eye Clinic from June, 1953 to January, 1955. All but 11 percent occurred in industrial workers and a hammer was implicated in 78.5 of industrial injuries; 64 percent of foreign bodies were in the posterior segment of the eyeball and 21 percent in the anterior chamber. The improvement in results over those in other series are ascribed to improved surgical and diagnostic techniques, the use of antibiotics and the introduction of new instruments for extraction of nonmagnetic intraocular foreign bodies. (3 tables, 6 references) Sylvan Brandon.

Miller, H. A. **Retinal burns from atomic flash.** Ann. d'ocul. **190**:747-754, Oct., 1957.

The intensity of a retinal atomic flash burn is modified by three factors: the filtration and dispersion of rays by the atmosphere, the pupillary diameter and the lid closure reflex. The author then abstracts the data of Byrnes and Brown on the ophthalmoscopic appearance of retinal burns in rabbits and in man. The usual lesion is a central chorioretinitis similar to that seen in eclipse scotomata. (7 references) David Shoch.

Nécsei, P. **Attempts to prevent damages to metal foreign bodies penetrating into the eye.** Szemescet **1**:28-34, 1957.

The author provides experimental evidence that the endophthalmitis of siderosis or chalcosis can be prevented by the use of high frequency induction current. Gyula Lugossy.

Piffkó, P. **Bee's sting in the cornea.** Szemeszet **1**:43-44, 1957.

In an intralamellar corneal abscess due to bee's bite the sting was found. Removal of the sting resulted in full restoration with complete visual acuity. Gyula Lugossy.

18

SYSTEMIC DISEASE AND PARASITES

Amendola, F. and Saverio Blois, A. **Ocular manifestations of infectious tropical diseases of unknown or poorly defined etiology.** Arq. brasil. de oftal. **20**:67-74, 1957.

The Brazilian variety of pemphigus foliaceus differs from the type seen in this country in that it affects children and adolescents more often than adults, has a more benign course, and has abortive types. When there are eye complications, it is diagnosed as ocular pemphigus foliaceous. The corneal lesions are superficial bullae which rupture and are followed by ulcers and pannus. Degenerative changes may be found in the iris with pigmentary changes. Intumescence of lens fibers may develop and is followed by hydropic degeneration and opacification. Retrobulbar neuritis and alterations in the retinal vessels have also been observed. It is important from a prognostic standpoint to differentiate ocular pemphigus foliaceus from ocular pemphigus vulgaris in which lesions of the eye are more severe.

Trachoma is discussed in its four stages. Its etiology is in doubt; it is

possibly due to a virus of large size similar to that of psittacosis or lymphogranuloma inguinale. Ocular findings in rheumatic fever are edema of the eyelids, uveitis (more often observed in the chronic form of the disease), episcleritis or tenonitis. The uveitis is usually plastic in type and involves the entire uveal tract and is believed to be an allergic reaction to streptococcus. Rheumatoid arthritis generally has the same eye manifestations as does rheumatic fever. In addition, blepharitis and Sjøgren's syndrome may be observed. In erythema nodosum inflammation of the iris or ciliary body may occur. James W. Brennan.

Bogatova, A. **An ascarid under the conjunctiva.** Vestnik oftal. 3:49, May-June, 1957.

In the right eye of a woman, aged 38 years, the conjunctiva was incised and an ascarid 9 mm. by 0.5 mm. was extracted. Santonim was given to the patient and three young worms came out in the feces. Olga Sitchevska.

Charlin, C. **Ocular manifestations in juvenile rheumatoid arthritis.** Arch. chil. de oftal. 14:58-62, Jan.-June, 1957.

The author mentions the three most frequent ocular complications of the juvenile rheumatoid arthritis in its two forms: Stills disease and the deforming and ankylosing rheumatisms of infancy: chronic, torpid iridocyclitis, complicated cataract as a result of the iridocyclitis and band opacity of the cornea, which makes the finding of the cataract many times rather difficult. Sometimes glaucoma can also be found, but the ocular hypotony is a more frequent finding.

The author then gives a brief summary of a patient, seven years of age, with all three of the ocular complication. He advises atropine and cortisone as local treatments for the iridocyclitis in its early stages. Serious consideration must be given to corneal transplants and cataract extractions in the later complications. (2 figures, 3 references) Walter Mayer.

Corviniano Rodriguez. **A case of congenital toxoplasmosis.** Arch. Soc. oftal. hispano-am. 17:476-483, May, 1957.

This is a detailed report of a case of congenital toxoplasmosis in a girl, four months old. She had hydrocephalus, retinochoroidal lesions, intracraneal calcium formations, and convulsions. (10 figures) Ray K. Daily.

Cuendet, Madeleine. **Ocular care in patients with tetanus curarization.** Schweiz. med. Wchschr. 87:1194-1195, Sept. 21, 1957.

Lagophthalmus is one of the side reactions in the therapy of severe tetanus with curare. The cornea must immediately be protected from exposure keratitis: The use of fibrin film, glass occlusion, or blepharorraphy may be used as indicated in each individual case. (5 references) Irwin E. Gaynon.

Duke-Elder, S. and Maurice, D. M. **Symbols of ocular dynamics.** Brit. J. Ophth. 41:702-703, Nov., 1957.

A system of symbols for the various constants used in studies of intraocular dynamics was developed and circulated to workers in the field. It found ready acceptance. Morris Kaplan.

Espildora-Luque, C. **Unilateral post-hemorrhagic blindness.** Arch. chil. de oftal. 14:53-55, Jan.-June, 1957.

The author mentions the rarity with which blindness, usually bilateral, follows hemorrhage, generally medical hemorrhage which usually is not profuse but persistent or repeated. It has not been explained why this type of blindness is not more frequent. Usually it is a sequel of many gastric and uterine hemorrhages.

Usually the blindness appears from the third to the seventh day after the hemorrhage. Ophthalmoscopically there is ischemia of the retina, which is sometimes associated with retinal edema, palor of the disc and arterioles which are filiform. If fields can be taken a variety of signs may be found: scotoma of different size and position, hemianopsias, and concentric losses of field. The most frequent field change is a scotoma of the inferior half of the field, which is ascribed to the action of gravity, as any lack of blood will be more manifest in the upper portions of the retina.

The author then describes a patient who had a profuse hemorrhage after the draining of a maxillary sinus; difficulty of vision in one eye was noted four days later. The upper portion of the retina was ischemic. Recovery was fairly good.

Walter Mayer.

Goodman, G., von Sallmann, L. and Holland, M. G. **Ocular manifestations of sickle-cell disease.** A.M.A. Arch. Ophth. **58**:655-682, Nov., 1957.

The ocular manifestations of sickle-cell anemia in five cases are reported. The changes consist primarily in 1. retinitis proliferans, 2. aneurysmal vascular dilatations, 3. arborizing vascular networks, 4. focal constriction, 5. dilatation, sheathing, and obstruction of arterioles and venules, 6. the development of chalk-white vessels, and 7. widespread retinal and vitreous hemorrhages. Patients with sickle-cell hemoglobin C disease are especially prone to ocular changes. It may be concluded from the above that vascular stasis and obstruction of both the arterial and venous systems are the basis for the ocular complications. (21 figures, 2 tables, 57 references) G. S. Tyner.

Jadassohn, W., Franceschetti, A., and Golay, M. **Cutaneous reaction in a case of aphthous uveitis with recurrent hypopyon (Behçet's syndrome).** Schweiz. med. Wchschr. **87**:1188, Sept. 21, 1957.

An antigen was prepared from a nonulcerated lesion of the scrotum in a case of Behçet's disease. This antigen gave a tuberculin type of reaction on intradermal testing when used on the patient, but was negative in three control cases. (3 references) Irwin E. Gaynon.

Leishman, Robert. **The eye in general vascular disease. Hypertension and arteriosclerosis.** Brit. J. Ophth. **41**:641-701, Nov., 1957.

Involutionary sclerosis may be assumed to be a normal concomitant of old age and may occur prematurely in young people with or without increased blood pressure. Hypertension means a persistent elevation of both the systolic and diastolic levels of blood pressure and is the result of the increased peripheral resistance offered by widespread diffuse arteriolar hypertonus. If vessels are significantly affected by involutionary sclerosis, the effective hypertonus is reduced and these vessels are then protected from serious elevation of diastolic pressure. The balance or lack of balance between these two factors results in significant changes in the vessel walls which can be observed ophthalmoscopically and from these changes a clinical classification of vessel alterations may be made. Seven categories are suggested:

1. Involutionary sclerosis occurs universally after the age of 60 years and may be considered normal. This is associated with a rise in systolic pressure without an elevation of the diastolic. The retinal arteries are straight and narrow, the veins look normal.

2. Involutionary sclerosis with hypertension occurs when the arterioles are already in "defense sclerosis"; they are dilated bright red vessels with variations in caliber. This is seen in elderly subjects with benign hypertension or in younger

subjects with manifestations of premature senility.

3. Advanced involutionary sclerosis with hypertension: here definite senile changes are seen. The arterioles are red, wide and tortuous with the smaller branches which are pale, straight and narrow. These patients may have few complaints although their diastolic pressure may reach 140 mm.

4. The normal fundus in youth with normal blood pressure readings.

5. Early hypertension in youthful vessels may be associated with acute glomerulonephritis. The arterioles are unduly straight and narrow with a pale blood column. The veins are not concealed at crossings but may be congested beyond the crossings. The fundus may resolve completely with the abolition of the hypertension.

6. Fulminating hypertension results from very severe hypertension acting upon relatively youthful arterioles which are undefended by fibrosis. Papilledema and retinopathy are usually well marked. The veins are concealed at the arterial crossings and the arteries are pale and narrow though variable in caliber and may become quite tortuous. White thread-like obliterated arterioles are often noted. Small, round, hard-edged exudates are numerous near the disc and the macula, and cotton-wool exudates are scattered over the fundus. Small hemorrhages may be numerous and larger ones also occur. This lesion probably results from focal necrosis in the arteriolar wall of the vessels in the eye and other organs. It occurs mostly in younger subjects who note failing vision; the fundus picture is usually well established at the first examination when the diastolic pressure may be 160.

7. Severe hypertension with reactive sclerosis. A defensive fibrosis has begun and the degenerative changes are considerably less devastating. The disc is normal, the veins are obstructed by the crossing arteries and are congested distal to the crossing. Fibrosis, hyperplasia and hypertonus may be observed in the same vessel. The great majority of middle-aged people with hypertension fall into this group and often reach a state of benign hypertension because of the reactive fibrosis. This fibrotic reaction and atheromatous changes in the vessel walls may result in a partial or total occlusion of the vessel.

Secondary degenerative changes develop in the retina and are made manifest by hemorrhage and exudates. The retinopathies differ in appearance when caused by different etiologic agents and have been classified in five groups: 1. arteriosclerotic, 2. renal, 3. toxemic retinopathy of pregnancy, 4. malignant hypertensive, and 5. diabetic retinopathy.

The author points out that the presence of arteriolar fibrosis is a contraindication to extreme hypotensive therapy. The diastolic pressure must be maintained near 120 mm. Hg which allows time for further protection by replacement fibrosis. Inactivity in bed should be avoided and moderate activity with a minimal dose of hypotensive drugs should be maintained. The superb photographs of fundus and histologic preparations are invaluable. (63 figures, 5 references)

Morris Kaplan.

Levitt, Jesse M. **The oculoglandular form of cat-scratch disease.** J.A.M.A. 165: 1955-1956, Dec. 14, 1957.

Cat-scratch disease involving the eye takes the form of oculoglandular symptom complex or Parinaud's syndrome. The skin antigen test is necessary for diagnosis. The case reported responded to gantrisin. It is quite possible that the mycotics usually reported as the etiological factor are secondary invaders. The cat-scratch skin-antigen test should be done in all cases of Parinaud's syndrome. (2 figures, 9 references) Irwin E. Gaynon.

Matsuyama, M. and Kashiwai, T. **Ocular symptoms of encephalitis epidemica japonica seen in Okayama in 1956.** Acta Soc. Ophth. Japan **61**:1718-1725, Sept., 1957.

This is a statistical study of 96 cases. Among ocular symptoms are the disturbance in the pupillary reflex, change in the pupillary form, paresis of accommodation, ptosis, lagophthalmus, corneal opacities, and such fundus changes as dilatation of retinal vessels, hyperemia or pallor of the disc, and retinal hemorrhage. The symptoms appeared in the acute stage and disappeared in the convalescent stage. In general, however, the ocular symptoms were slighter than in Economo's encephalitis. (4 tables, 31 references)

Yukihiko Mitsui.

de Poli, A., Montsori, W., and Pietri, P. **Ophthalmologic aspects of chronic peripheral obliterating arteriopathies.** Arch. d'opht. **17**:455,464, 1957.

The authors note the recent clinical and experimental research on peripheral arteriopathies and stress the importance of the classification and differential diagnosis of the various forms. They studied the retinal vessels in 140 cases of peripheral arterial disease and noted striking differences between cases with arteriosclerotic disease and cases with thrombotic vascular lesions. In the thromboangiotic cases vascular sclerosis and venous dilatation were minimal whereas in the arteriosclerotic arteritis cases the retinal vessels showed major involvement with periarteritis and sclerosis of both veins and arterioles. Arteriovenous crossing signs were particularly prominent. The authors stress the importance of ophthalmoscopic examination as an aid in classifying cases and in determining the medication of choice. (2 figures, 5 tables, 21 references)

P. Thygeson.

Smith, Redmond. **Ocular changes in diabetes mellitus.** Brit. J. Cl. Pract. **11**: 495-501, July, 1957.

The ocular complications of diabetes are listed topographically. The most detailed discussion is on diabetic retinopathy, wherein the histopathology and clinical picture are evaluated in the light of modern thought. Diabetic retrobulbar neuritis is mentioned, as is the increased susceptibility of diabetics to tobacco amblyopia. Refractive changes due to lens variations and diabetic cataracts are discussed, as well as senile cataracts in diabetics. Changes in the iris such as tabetic type pupillary changes, rubeosis iridis, diabetic uveitis, and diabetic swelling of the pigment cells are mentioned. The possibilities of specific changes in the conjunctival vessels are discussed. Other complications are muscle palsies, subjective visual disturbances due to hypoglycemia, decrease in intraocular pressure in diabetic coma, and glaucoma due to angle neovascularization. (12 figures, 17 references)

Harry Horwich.

Wright, W. H. **A summary of the newer knowledge of toxoplasmosis.** Am. J. Clin. Path. **28**:1-17, July, 1957.

Evidence accumulated by the author during the past eight years indicates that toxoplasmosis is more prevalent in warm, moist areas than in cold or hot, dry climates. Lower animals are assumed to be reservoir hosts for toxoplasma and man acquires this infection from them. Two types of toxoplasmosis are recognized; the congenital type and the acquired type. One form of the congenital type is characterized by the following tetrad of symptoms: chorioretinitis, sometimes with nystagmus and microphthalmia; microcephaly or hydrocephaly; convulsions; and cerebral calcifications. In some cases, chronic clinical signs, usually chorioreti-

nitis, may be noted as long as a year after an apparent normal birth. In the acute stages of congenital toxoplasmosis the following manifestations may be noted; fever, icterus, generalized maculopapular rash, splenomegaly, hepatomegaly, pneumonitis, and myocarditis. Acquired toxoplasmosis is classified into the exanthematic, cerebrospinal, ophthalmic, and lymphadenopathic forms. Literature is cited by the author which indicates that toxoplasmosis is a common cause of granulomatous uveitis. Serologic tests (methylene blue and complement-fixation tests) should be performed in all suspected cases of toxoplasmosis. The only sure method of diagnosis is by isolation of toxoplasma from the infected individual. The drugs which have shown the most promise are a combination of pyrimethamine (Daraprim) and the sulfonamides. It must be remembered that pyrimethamine may cause various blood changes. (8 tables, 35 references)

Ted Suie.

Zigas, V. and Gajdusek, D. C. **Kuru: clinical study of a new syndrome resembling paralysis agitans in natives of the Eastern Highlands of Australian New Guinea.** M. J. Australia **2**:745-754, Nov. 23, 1957.

The authors investigated a new syndrome causing widespread neuronal degeneration with severe effects in the cerebellum and extrapyramidal system. The disease appears to be localised to one tribal area but no cause nor specific treatment has been found. Convergent squint appears late in the illness but otherwise the eyes reveal no abnormalities. When severe tremors are present the patient has difficulty in maintaining a fixed gaze. Nystagmus is absent and the pupils react normally. There is no papilledema, optic atrophy or retinopathy. There is no greenish-yellow pigmentation at the sclerocorneal border (Kayser-Fleischer ring). (10 figures, 10 references)

Ronald Lowe.

19

CONGENITAL DEFORMITIES, HEREDITY

Cuendet, J. F. **Heredity counselling in eye diseases.** Eugenics Quart. **4**:139-147, Sept., 1957.

The author discusses the numerous hereditary ocular characteristics and lesions which are detectable because of the clarity of the media of the eye. This makes it easier for the physician to counsel persons with a particular disease who consider having a child.

Prognostic tables of risk are presented for sex-linked, recessive, and dominant autosomal eye disease under various conditions of consanguinity and the number of antecedents involved. An alphabetic list is appended with eye diseases characterized by their mode of inheritance and estimated accuracy of genetic prognosis. (4 tables, 5 references) Irwin J. Cohen.

Delaloye, J. and Stucchi, C. **Keratoconus, cataract and mongolism.** Schweiz. med. Wchschr. **87**:1202-1203, Sept. 21, 1957.

The authors report a case of mongolian idiocy, bilateral keratoconus, and cataract in a 42-year-old woman with oligophrenia. It is thought that a general endocrine dysfunction is responsible for this syndrome. (2 figures, 17 references)

Irwin E. Gaynon.

Rintelen, F. and Stauffenegger, U. **The pathogenesis of pseudophakia fibrosa.** Arch. f. Ophth. **159**:411-419, 1957.

The authors describe histopathologic preparations of an eye with numerous congenital anomalies. Persistent hyaloid membrane with partial persistence of the primary vitreous body is occasionally a

result of retarded development with which hypertrophic manifestations and disturbances of development of the lens may be associated. This may occur as an example of the teratological principle to which Werthemann called attention, namely that one organ may be influenced in its development by another organ. In this case retarded development, that is persistent primitive vitreous, was effective in the development of a secondary anomaly of the lens in the form of cataract, secondary aphakia or pseudophakia fibrosa. (6 figures, 10 references)

F. H. Haessler.

Waardenburg, P. J. **Interocular hyperplasia with dystopia of the medial canthi, blepharophimosis, iridocutaneous dyschromia, and auditory dysplasia.** Acta ophth. 35:311-324, 1957.

The author describes his study of this syndrome which comprises a hyperplastic development of the interocular region with bilateral congenital deafness, leading to mutism, or unilateral congenital deafness, and pigment anomalies. The hyperplasia of the interocular region expresses itself in a large nasal bridge, overdevelopment of the medial portion of the eyebrows, excessive interpupillary distance, dystopia lateroversa of the inner canthus and lacrimal puncti, and blepharophimosis. The pigment disturbances may manifest themselves as gray locks, partial or total heterochromia iridis, bilateral hypoplasia of the stroma pigment, and depigmented cutaneous areas. All manifestations of this syndrome may be encountered as isolated dominant hereditary defects with the exception of deaf-mutism which is inherited sporadically as a recessive. In 1951 the author reported seven Dutch families, in which the syndrome was inherited as a dominant. The dystopia lateroversa of the canthi was found in 99 percent of the cases; the other anomales were less frequent. The penetrance of the heterochromia was 25 percent, of deaf-mutism 20 percent, and of white locks 17 percent. The literature subsequent to his report is reviewed, and the contribution of further cases in Holland and in other countries described. Combinations of anomalies similar to this syndrome have been described in animals, especially in cats and dogs. The various phases related to heredity and to the genesis of isolated anomalies are discussed. Particularly interesting is the occurrence of this syndrome in negroes, who then may have blue eyes, white hairlocks and deaf-mutism. (6 figures, 3 pedigrees, 11 references) Ray K. Daily.

20

HYGIENE, SOCIOLOGY, EDUCATION, AND HISTORY

Beltran, S. and Araya, A. **Ocular causes of disability.** Arch. chil. de oftal. 14:42-48, Jan.-June, 1957.

The authors discuss the regulation of compensation for ocular disabilities in Chile and recommend that the compensation laws be amended.

Walter Mayer.

Guerra, Paolo. **Racial and regional differences in the epidemiology and clinical manifestations of trachoma.** Wld. Hlth. Org. Bull. 16:1038-1041, 1957.

Volunteers in Eritrea were inoculated with trachoma II and the results confirmed the author's previous observations in Ethiopia on the variation in susceptibility of various racial and ethnic groups. The study also indicated that the onset of trachoma is normally chronic. (4 tables, 1 reference) Edward U. Murphy.

Mann, Ida. **Report on ophthalmic findings in Warburton Range Natives of Central Australia.** M.J. Australia 2:610-612, Oct. 26, 1957.

An account is given of the ophthalmic

investigations of 438 caste and full-blooded natives. Trachoma was the only ocular disease of importance. Trachoma was widespread but not severe. The infection rate of the population was 77 percent of which 62 percent were in the active and infectious stage. There were no ocular signs of malnutrition. The following diseases, although common in whites, were not encountered: styes and marginal blepharitis, mucopurulent conjunctivitis, myopia, convergent squint, glaucoma, water-cleft cataract and color blindness. (4 tables, 3 references) Ronald Lowe.

Orlowski, Witold J. **Polish ophthalmological bibliography 1956.** Klinika Oczna 27:301-304, 1957.

The author lists the titles of 76 ophthalmologic reports which appeared in the Polish non-ophthalmological medical literature for the year 1956 and four additional titles which appeared earlier but were missed in previous surveys.

Sylvan Brandon.

Pallares, J. **A brief ophthalmologic excursion in France.** Arch. Soc. oftal. hispano-am. 17:505-532, May, 1957.

This is a very interesting diary of the author's visits to the ophthalmologic clinics of Bordeau, Nantes, Paris and Toulouse. (2 references) Ray K. Daily.

Siniscal, A. A. **Epidemiological and clinical aspects of trachoma in the U.S.A.** Wld. Hlth. Org. Bull. 16:1047-1050, 1957.

The incidence of trachoma has declined remarkably in the United States in the past twenty years, both in the Indian and white populations. The severity of the infection is less and the number of operations done for late complications has greatly decreased. The trachoma control programs are changing from treatment of active cases to intensive preventive medicine and case finding in the field. There is still a group of older patients with cicatricial complications from active infection 25 to 50 years ago. (1 reference)

Edward U. Murphy.

Skydsgaard, Henning. **The eugenic problems in the prevention of blindness.** Acta ophth. 35:325-335, 1957.

The author describes the provision for the supervision of the blind in Denmark and reports the data accumulated over a period of 18 years in the Eye Clinic of the Institutes for the Blind. The Eye Clinic works in collaboration with the University Institute for Human Genetics which has reports on all people cared for by the National Welfare Service for the Blind. In addition to endeavoring to gather research data which would add to the knowledge on the transmission of each disease, the clinic provides advice concerning the eugenic prognosis of potential offspring of people about to be married, the eugenic indications for induced abortion during pregnancy, and the question of sterilization. It provides guidance to parents of blind children, and examinations for children of blind parents. The material, which consists of 473 cases, is analyzed as to the category of diseases, the quantitative distribution of the eye diseases, the eugenic prognosis of parental combinations, the eugenic indications for the 40 induced abortions, the indications for the 15 cases of sterilization, and the advice to normal-sighted parents of blind children. This field of work, which the author calls social ophthalmology, requires great tact and ability to base advice on careful objective evaluation of the clinical, social, and human circumstance of each case. (8 tables) Ray. K. Daily.

NEWS ITEMS

EDITED BY DONALD J. LYLE, M.D.
411 Oak Street, Cincinnati 19, Ohio

News items should reach the editor by the 10th of the month. For adequate publicity, notices of postgraduate courses and meetings should be received three months in advance.

DEATHS

Dr. Vesse Reeves Hurst, Longview, Texas, died December 7, 1957, aged 70 years.

Dr. Oscar Lloyd Veach, Sheridan, Wyoming, died November 10, 1957, aged 73 years.

ANNOUNCEMENTS

COURSE IN SLITLAMP BIOMICROSCOPY

The Committee on Postgraduate Education of the Montefiore Hospital, Pittsburgh, announces an advanced course in slitlamp biomicroscopy of the living eye combined with additional courses in slitlamp microscopy, gonioscopy, deep vitreous and retinal biomicroscopy, indirect ophthalmoscopy, surgery of intraocular foreign bodies, and retinal detachment surgery, to be given by the Montefiore Department of Ophthalmology under the direction of Dr. Harvey E. Thorpe and associates. These courses will be given at the Montefiore Hospital, for four days, May 5th to 8th, inclusive, from 8:00 A.M. to 4:00 P.M.

Guest speakers will be Dr. Robert J. Masters, Indianapolis; and Mr. Lee Allen, Iowa City, Iowa.

For further information write to:
A. McNabb
Secretary of Postgraduate Ophthalmic Courses
206 Iroquois Building
Pittsburgh 13, Pennsylvania

ORTHOPTIC EXAMINATIONS

The annual examination of orthoptic technicians by the American Orthoptic Council will be conducted in August and October, 1958.

The written examination will be nonassembled and will take place on Thursday, August 21st, in certain assigned cities and will be proctored by designated ophthalmologists.

The oral and practical examinations will be on Saturday, October 11th, in Chicago just preceding the meeting of the American Academy of Ophthalmology and Otolaryngology.

Application for examination will be received by the office of chairman of examinations:
Dr. Frank D. Costenbader
1605 22nd Street, N.W.
Washington 8, D.C.

Applications, which must be accompanied by the examination fee of $30.00, will not be accepted after July 1, 1958.

BARRAQUER INSTITUTE COURSE

The Instituto Barraquer, Laforja 88, Barcelona, Spain, announces the second international course in ophthalmology to be held at the Institute from September 16 to October 1, 1958, under the direction of Prof. Ignacio Barraquer.

Comprising the program will be a number of papers and symposia on opthalmic problems. There will be surgical sessions and the showing of moving picture films. On Wednesday, September 17th, the following papers will be presented: "The mesopic visual field," G. E. Jayle; "The role of tonsils in certain ocular affections," J. Legrand; "Instrumentation in ophthalmic surgery," H. Katzin; "Biomicroscopy," A. Busacca; "The optic radiation in man," J. Malbran; "Corneal dehydration." B. Carreras Matas.

The subject of the symposium to be held on Thursday, September 18th, will be "Glaucoma." The chairman will be B. Boyd. On the panel will be P. A. Chandler, D. Reavitz, A. Moreu, A. Posner, A. B. Rizzuti, and B. Strampelli. Those taking part in the discussion will be J. Barraquer, B. Carreras Matas, J. François, E. Malbran, A. E. Maumenee, A. Muinos, A. Salleras, M. Sanchez Salorio, A. Schlossman, J. M. Simón, R. Troutman, and D. Vail.

Mr. B. W. Rycroft will discuss the "Surgery of congenital ptosis," on Friday, September 19th. The symposium on that date will be on "Corneal grafting," and A. Salleras will be chairman. On the panel will be M. Amsler, J. Barraquer, J. Legrand, R. T. Paton, F. Ridley, H. Rocha, and B. W. Rycroft. The following will take part in the discussion: G. B. Bietti, J. François, G. E. Jayle, G. B. Kara, H. Katzin, J. H. King, A. E. Maumenee, E. Schreck, and B. Strampelli.

On Saturday, September 20th, G. Favaloro will speak on "Optical centers and tracts of the hypothalamus"; J. François, "The importance of electroretinography in the differential diagnosis of tapetoretinal degenerations"; M. Amsler, "A study of the aqueous humor."

The symposium on "Surgery of the crystalline lens" is scheduled for Monday, September 22nd, with R. Troutman as chairman, and I. Barraquer, J. Barraquer, G. B. Karam, A. Maumenee, A. Posner, A. Salleras, and D. Vail on the panel. The discussors will be B. Boyd, P. A. Chandler, O. Ferrer, J. François, H. Katzin, J. Malbran, G. Rama, H. Ridley, B. W. Rycroft, M. Sanchez Salerio, E. Schreck, and D. M. Shafer.

On Tuesday, September 23rd, H. Ridley will speak on "Metazoan diseases of the eye"; L. Maggiore, "Thrombosis of the retinal veins in hematovascular pathology"; G. Cucco, "Pathology of the

retinal veins." The symposium on Wednesday, September 24th, will be "plastic lenses," with J. Barraquer as chairman. H. Dannheim, O. Ferrer, W. S. Reese, H. Ridley, J. Scharf, H. Schreck and B. Stampelli will be on the panel, and the discussors will be G. B. Bietti, B. Boyd, A. Huber, G. B. Kara, H. Katzin, J. H. King, J. Legrand, G. Rama, A. Salleras, C. D. Shapland, and R. Troutman.

Presenting a paper on Thursday, September 25th, will be L. Maggiore, "Color and chromatic vision in normal and pathologic conditions." "Retinal detachment" will be the subject of the symposium on Friday, September 26th, with C. D. Shapland as chairman. On the panel will be S. Forni, G. Meyer-Schwickerath, P. McG. Moffatt, A. Muiños, H. Pierce, and D. Shafer. Discussors will be M. Amsler, H. Arruga, A. Bangerter, G. Bonamour, B. Boyd, O. Ferrer, J. Legrand, L. Maggiore, J. Malbran, B. Strampelli, and D. Vail.

Papers on Saturday, September 27th, will be read by G. Bonamour, "Some malformations of the papilla and their clinical interest"; A. Bangerter, "Diagnosis and treatment of alterations of the lacrimal apparatus"; G. B. Bietti, "Recent advances in the treatment of some ocular conditions."

On Monday, September 29th, there will be a symposium on "Strabismus," with A. Huber as chairman and H. M. Burian, C. Cueppers, A. Huber, E. Hugonnier, K. Lyle, A. Muiños, and A. Schlossman on the panel. Discussors will be A. Bangerter, G. B. Bietti, P. A. Chandler, E. Melbran, J. Malbran, G. Rama, A. Salleras, and G. Sevrin.

Papers to be presented on Tuesday, September 30th, will be "Nontypical forms of corneal virosis," M. Sanchez-Salorio; "Physiologic problems of visual acuity," A. Pinero Carrion; "Orbital tumors," R. Bartolozzi.

The closing session will be on Wednesday, October 1st.

In addition to the scientific program, a most interesting recreational program has been planned. There will be excursions, bullfights, demonstrations of Andalusian folklore, and so forth. The official languages of the course will be Spanish, French, English, German, and Italian. There will be simultaneous translations from and into each of these tongues. For registration blanks and programs write to:

Secretary
Instituto Barraquer
Laforja 88
Barcelona, Spain

Courses at Mount Sinai

Columbia University is sponsoring the following courses at The Mount Sinai Hospital in New York:

1. Histopathology of the eye, to be given during April, 1958, by Dr. Joseph Laval, Dr. David Wexler, Dr. Abraham Kornzweig, and Dr. Alan Barnert.

2. Gonioscopy and tonography, by Dr. Sylvan Bloomfield and Dr. Jules Yasuna, during October, 1958.

3. The Schepens' binocular indirect ophthalmoscope and its use, and a discussion of the indications for the various operations for the cure of detached retina. This course will be given by Dr. William Toll and Dr. David Silver during December, 1958.

4. Ophthalmoscopy for general practitioners and pediatricians, by Dr. Jacob Goldsmith, Dr. Robert Coles, and Dr. Robert Sturman during November, 1958.

For complete information address:

The Registrar for Post-Graduate Instruction
The Mount Sanai Hospital
1 East 100th Street
New York 29, New York.

Essay Contest

The Instituto Barraquer, Laforja 88, Barcelona, Spain, announces a contest for papers on ophthalmic subjects. Three main prizes of 5,000, 3,000, and 1,000 pesetas will be awarded, as well as several prizes of 500 pesetas. The rules and conditions of the contest are:

1. Any physician under the age of 40 years, Spanish or of any other nationality, may compete. He may or may not be a member of the institute; however, members of the Board of Rectors are not eligible.

2. The contest ends December 31, 1958.

3. The papers may be written in the author's own language and must be accompanied by an extensive summary (about 500 words) which will be used for translation.

4. All papers must be typewritten, double spaced, on one side of the paper only. The page number should be in the upper right hand corner and one word identifying the manuscript in the upper left corner. Papers may be accompanied by illustrations, tables, and so forth on cardboard, with the corresponding page number and identifying word.

5. Accompanying the manuscript should be a small envelope with the author's full name and address sealed within; written on the outside should be the identifying word which has been placed in the manuscript.

6. The Board of Rectors and the Editorial Board will act as judges. Their decisions will be final and without appeal.

7. Manuscripts which receive no prize will be destroyed after three months unless the author requests its return by giving permission in writing to open the envelope that contains his name and address.

Harvard basic science course

The Harvard Medical School announces courses for graduates in basic sciences in ophthalmology beginning September 22, 1958, and continuing through January 17, 1959. The courses and instructors are:

Anatomy—Russell LeG. Carpenter, Ph.D.
Biochemistry and histochemistry—Jin H. Kinoshita, M.D., David G. Cogan, M.D., and associates

Microbiology and aseptic technique—Henry F. Allen, M.D.
Neuroanatomy—David D. Donaldson, M.D.
Neuro-ophthalmology—Richard B. Pippitt, M.D., and associates
Pathology and histopathology—Taylor R. Smith, M.D., and associates
Physiology and toxicology—Alfred W. Scott, M.D., W. Morton Grant, M.D., Endre A. Balazs, M.D., George Wald, Ph.D.
Visual optics and physiology—Paul Boeder, Ph.D.

From January 19, 1959, to March 11, 1959, a course in the introduction to clinical ophthalmology will be given:

Refraction—Albert E. Sloane, M.D.
Ocular motility—Abraham Pollen, M.D.
Strabismus—Virgil G. Casten, M.D.
Biomicroscopy—William P. Beetham, M.D.
Glaucoma—Robert R. Trotter, M.D., Paul A. Chandler, M.D., W. Morton Grant, M.D.
Retinal detachment and indirect ophthalmoscopy—Charles L. Schepens, M.D., Robert J. Brockhurst, M.D., and associates
Uveitis, plastic surgery, cataract and miscellaneous topics—Staff members

For further information, write to:
Assistant Dean
Harvard Medical School
Courses for Graduates
25 Shattuck Street
Boston 15, Massachusetts

Miscellaneous

Wills meeting

Papers presented at the 10th annual clinical conference of the Wills Eye Hospital, Philadelphia, were: Uveitis symposium: "Etiologic role of streptococci," "Sarcoid and fungi in the etiology," "Nonspecific immunologic tests in diagnosis," "Advances in therapy," J. W. Hallett, T. Hedges, M. I. Wolkowicz, I. H.. Leopold, T. Sery, and Q. Feria.

"Recent experiments on aqueous humor formation," Harry Green; "The amplitude of cyclofusion," S. I. Askovitz; "Neuro-ophthalmologic evaluation of abducens paralysis," E. C. Schrader and N. S. Schlezinger; "The present status of orthoptics," William E. Krewson, 3rd; "Preplaced appositional sutures in cataract surgery (film)," Wilfred E. Fry; "Cataract extraction and a method of wound closure (film)," Howard F. Hill; "Anterior chamber lens implant for aphakia (with film)," Robert A. Brown.

"A double-pointed, tapered needle for conjunctival closure," Charles G. Steinmetz 3rd; "Leukemia with unilateral exophthalmos," Harold A. Hanno; "Teen-aged central retinal arterial occlusion," Harold A. Hanno and Robert C. Lee; "Ocular complications in multiple myelomatosis," Edward J. Donnelly; "Electrolysis of iris cyst (film)," Patrick J. Kennedy.

"Modified Toti-Mosher operation (film)," Kelvin A. Kasper; "Enfolding of sclera for retinal detachment (film)," J. S. Shipman and C. M. Luce; "Vitreous implants in retinal detachment surgery," W. H. Annesley and P. J. Kennedy; "Plastic repair of lid scars (film)," Gerard M. Shannon; "Ptosis surgery (film)," Edmund B. Spaeth.

During the first evening of the conference there was a joint meeting with the Section on Ophthalmology, College of Physicians of Philadelphia, at which Dr. Georgiana Dvorak Theobald, Oak Park, Illinois, was guest speaker. Dr. Paul A. Chandler, Boston, presented The Arthur J. Bedell Lecture; the subject of his address was "Problems in the diagnosis and management of lens reaction." Mr. John A. Diemand, president of the Board of Directors of City Trusts, Philadelphia, was the guest-of-honor of the conference.

Pittsburgh joint meeting

The Pittsburgh Ophthalmological Society and the Pittsburgh Otological Society recently held a joint meeting with the Southwestern Pennsylvania Chapter of the American College of Surgeons at Pittsburgh. Lorand V. Johnson, Cleveland, presented a paper on "Bullous keratopathy as a complication of cataract extraction: Pathologic and therapeutic considerations." Moderator for the panel discussion on "Surgery in the aged," was Samuel F. Marshall, Boston. Collaborators were Rupert B. Turnbull, Cleveland, Elmer Hess, Erie, Frank J. Gregg, Pittsburgh, and Francis Foldes, Pittsburgh.

New York alumni meeting

At the recent annual spring meeting of the alumni and staff of The New York Eye and Ear Infirmary, Brittain F. Payne acted as moderator for the symposium on "Ocular pathology"; and John R. Finlay, Bernard Roberts, John T. Simonton and S. L. Samuels served as panel. Milton L. Berliner was moderator for the symposium on "Retinal detachment surgery," with members of the panel being Alfred Weintraub, Bernard Kronenberg, Morton Rosenthal, and David Silver.

For the symposium on "Glaucoma," Willis S. Knighton was moderator; the panel being A. A. Cinotti, S. Goodstein, M. Cholst, and Jay G. Linn. Byron Smith moderated the symposium on "Ocular injuries." and on the panel were A. Russell Sherman, Irving Schwartz, Arno Town, and L. Schachne.

Elbryne G. Gill was moderator of the symposium on "Cataract surgery," the panel being J. Gordon Cole, Joseph H. Krug, Gerald Kara, and Benjamin Rosenthal. Webb Chamberlain acted at moderator for the symposium on "Motility." On the panel were Hunter H. Romaine, Paul McAlpine, Abraham Schlossman, and Samson Weingeist.

Courses presented during the meeting were: "Gonioscopy and tonography," A. A. Cinotti, S. Goodstein, and Vincent Carter; "Surgical approach to the orbit," John T. Simonton; "Corneal transplant," Jorge N. Buxton; "Electrophysiology,"

Jerry H. Jacobson; "Orthoptics," Hunter H. Romaine, Miss Jane Romanio, and John Leaman; "Indirect ophthalmoscopy," Morton Rosenthal.

"Macula function testing," Bruno S. Priestly and William Howard; "Radioactive isotopes in ophthalmology," Bernard Goldberg; "Biomicroscopy of the vitreous," Milton L. Berliner and Armand Violé; "Failures in ophthalmic surgery," Joseph H. Krug and J. Elliott Blaydes, Jr.; "Contact lens fitting," Blake Smith and Lois Peterson; "Anesthesia," J. Louis Heller and M. Livingstone; "Ocular photography," John Goeller; "Ocular pathology," John R. Finlay and E. Almeda.

Kansas postgraduate course

The University of Kansas School of Medicine, the Kansas City Society of Ophthalmology and Otolaryngology and the Kansas Medical Society presented at postgraduate course in ophthalmology at the University of Kansas Medical Center on April 7th, 8th, and 9th.

Guest instructors were Arthur J. Jampolsky, San Francisco; Frank W. Newell, Chicago; and R. Townley Paton, New York. Kansas University School of Medicine faculty included Larry L. Calkins, A. N. Lemoine, Jr., Charles M. Poser, Jesse D. Rising, James T. Robinson, Jr., and Dick H. Underwood.

Pittsburgh clinical meeting

On the ophthalmology program of the eighth clinical meeting of the Eye and Ear Hospital, Pittsburgh, Pennsylvania, were: "Retinal neovascularization," John C. Dunbar; "Treatment of Mooren's ulcer by corneal transplant," Philip C. Grana; "Intraocular pressure after pentothal anesthesia and retrobulbar anesthesia," William G. Everett, Edwin K. Vey, and Cornelius Y. Veenis. At the joint eye, ear, nose, and throat program, the "Re-evaluation of basic therapy in ophthalmology and otolaryngology" was discussed. Moderators were Robert J. Billings and George C. Schein. Collaborators were Frank M. Mateer, Horace M. Gezon, and Gerald P. Rodnan. The Hon. James Fulton, member of Congress, was the guest speaker at the meeting, and Dr. Daniel S. DeStio, president of the staff, was presiding officer.

Societies

Montana academy

At the recent midwinter meeting of the Montana Academy of Oto-Ophthalmology, at the Baxter Hotel, Bozeman, the following officers were elected: R. O. Lewis, Helena, president; R. M. Morgan, Helena, secretary-treasurer; F. H. Burton, Butte, president elect. The summer meeting will be held at Billings, Montana, on September 10th to 12th.

Nassau meeting

At the February meeting of the Nassau (county, New York) Ophthalmological Society, Dr. Hunter H. Romaine presented a paper on "Problems in ocular motility."

Pacific coast meeting

The annual meeting of the Pacific Coast Oto-Ophthalmological Society will be held at the Hotel Vancouver, Vancouver, British Columbia, on May 11th to 15th. The guest speakers will include: Thomas F. Dougherty, Victor Richards, Elton L. McCawley, Paul H. Holinger, Paul A. Chandler, and Vincent L. Rees. Collaborators on the symposium on "Maxillofacial trauma" are: Frank H. Wanamaker, Mark Gorney, Shirley Baron, Gordon McCoy, and Joseph Osterloh.

Personals

The list of honors published on January 1, 1958, contains the name of Sir Stewart Duke-Elder who was elevated to G.C.V.O. *The Lancet* of January 11, 1958, in a leader, "New Year Honours," has this to say about it:

"The particular reason for Sir Stewart Duke-Elder's promotion to be a Knight Grand Cross of the Victorian Order is presumably his services to the Royal Family, but these are of course only one manifestation of his remarkable services, through ophthalmology, to medicine."

Dr. Algernon B. Reese, clinical professor of ophthalmology, College of Physicians and Surgeons, Columbia University, New York, has been appointed to the Board of Scientific Counselors of the National Institute of Neurological Diseases and Blindness for a two-year term.

The Department of Ophthalmology of the New York University-Bellevue Medical Center has appointed Dr. Goodwin M. Breinin to the first Daniel B. Kirby professorship of research ophthalmology.

Mr. E. S. Perkins, F.R.C.S., reader in ophthalmology at the University of London and a surgeon at Moorfields, was a guest lecturer at the Boston City Hospital during January. The subject of his address was "Problems of tonometry."

The Section on Ophthalmology of the University of Louisville School of Medicine announces the appointment of Dr. Roderick Macdonald, Jr., to the position of assistant professor and executive director of the section, effective August 1, 1957. This full-time faculty appointment is a part of the expansion program in ophthalmology, both clinical and research, which is currently under way. Dr. Macdonald was formerly instructor in ophthalmology at Tulane University, New Orleans.

Urgent Request

The Uveitis Laboratory, University of California School of Medicine, San Francisco, is interested to obtain freshly enucleated eyes from patients with *all types of uveitis* and other endogenous inflammations. Attempts are being made to isolate etiologic agents from these eyes.

The eyes should *not* be fixed in preservatives or frozen, but placed in a sterile bottle, packaged, and shipped as quickly as possible. Please send specimens air express, special delivery, *collect*. Enclose history and findings and mark the package "Fresh Tissue Specimen—Rush."

A report of isolations of organisms and pathologic findings, including a slide, will be sent to the contributor. Credit will be given in any resulting publications if desired.

• • • •

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